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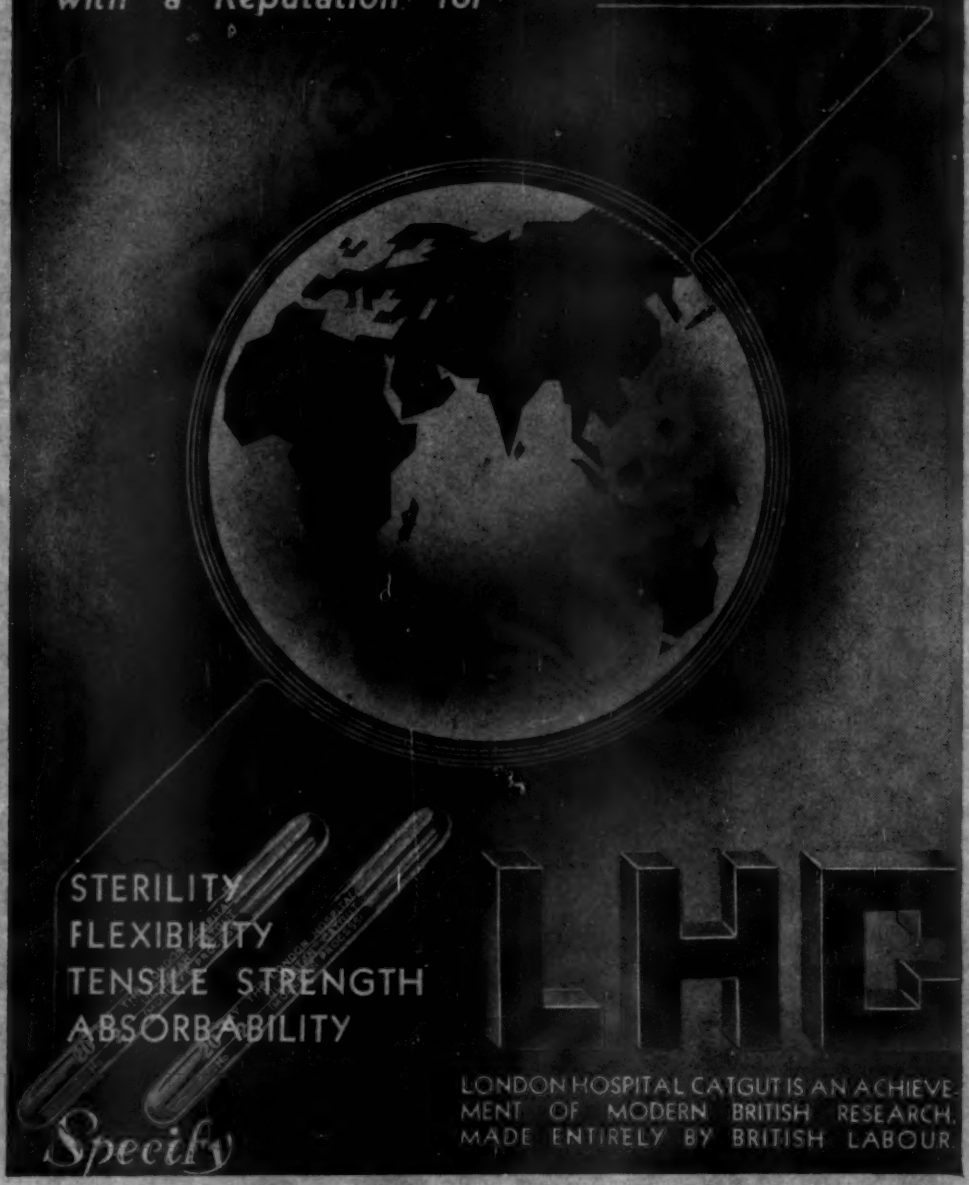

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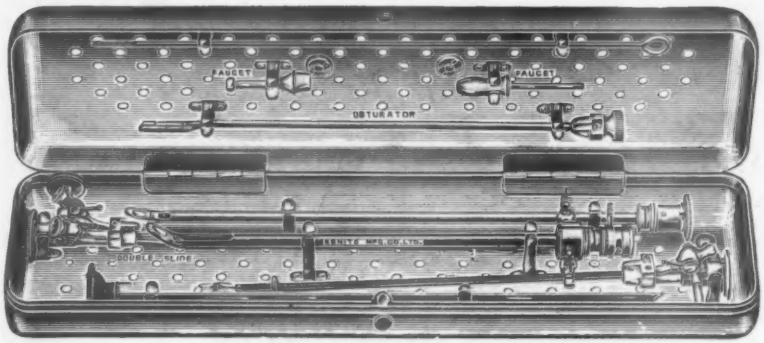

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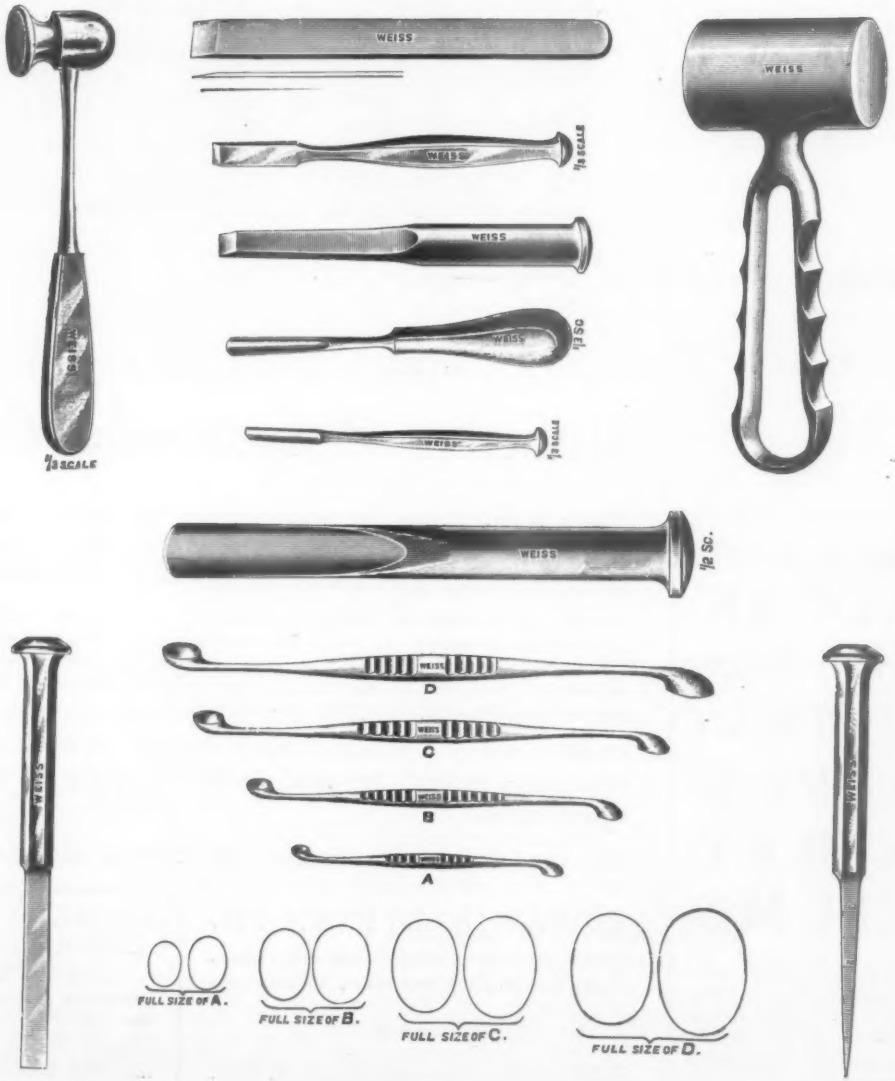

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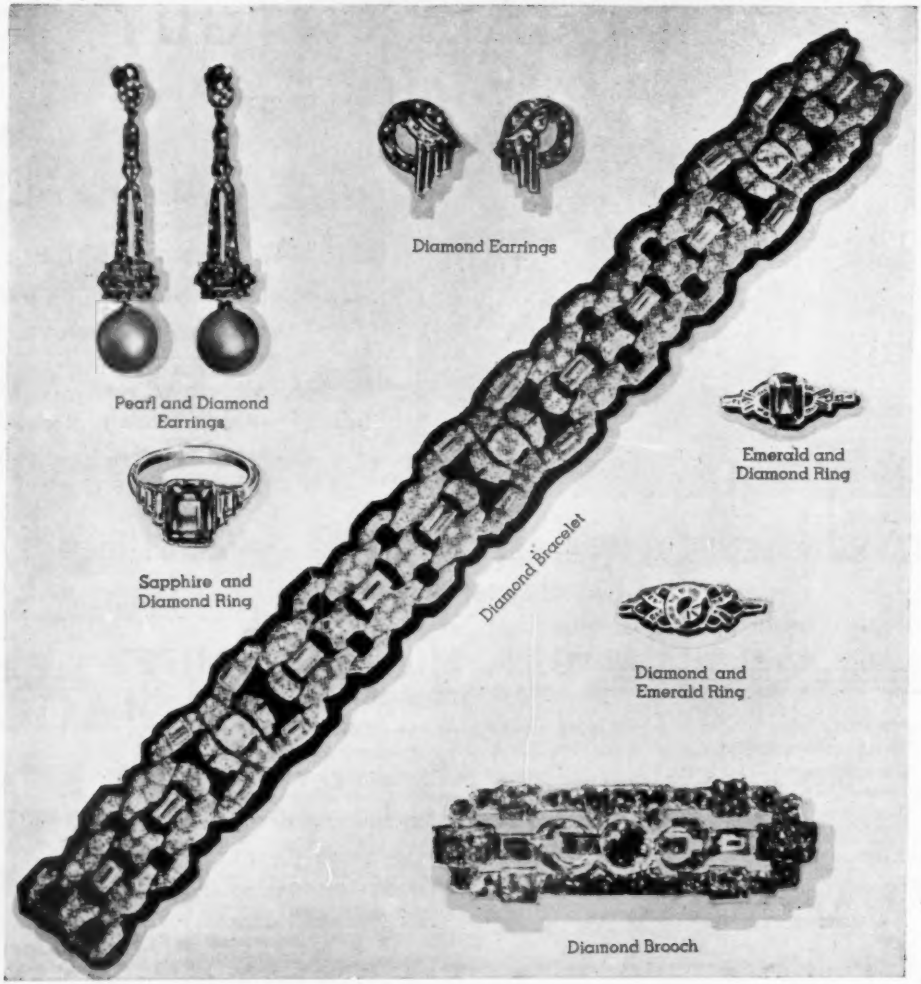

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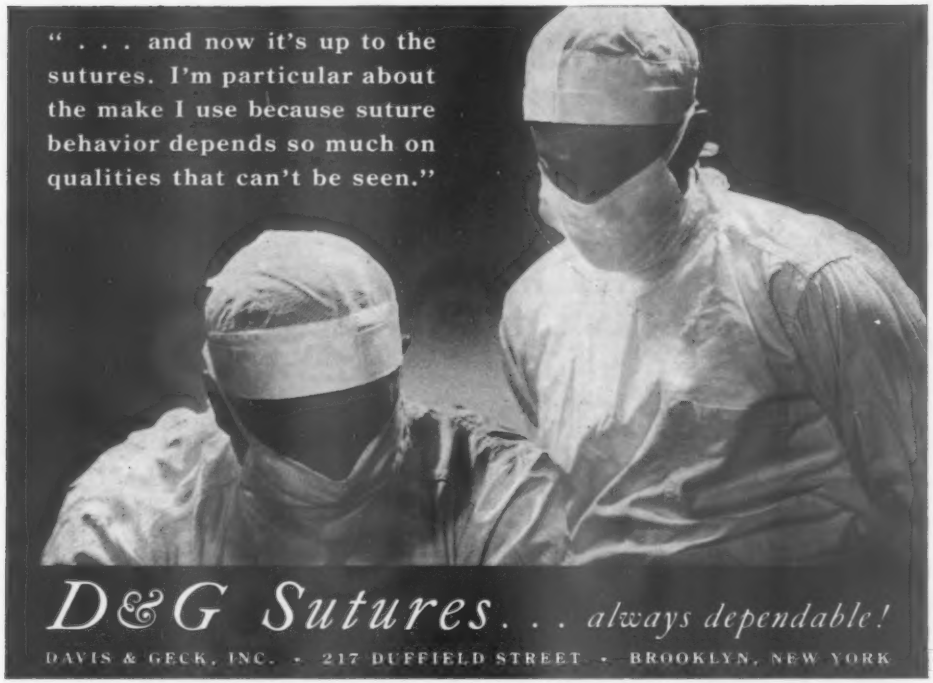
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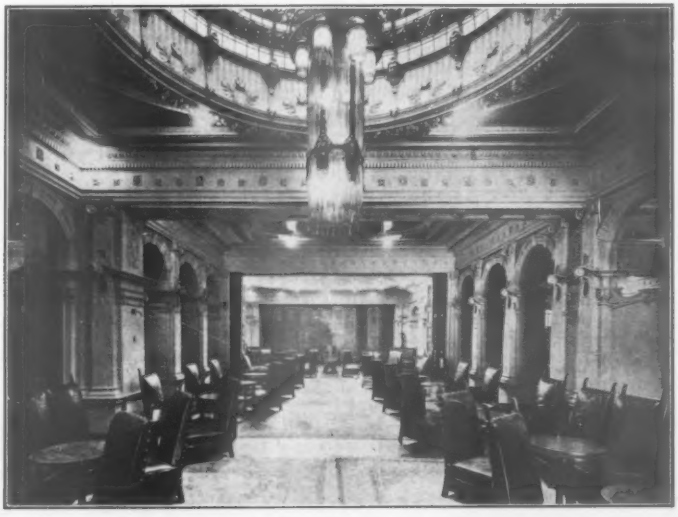

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(*Der Chirurg*, 15th May, 1936.)

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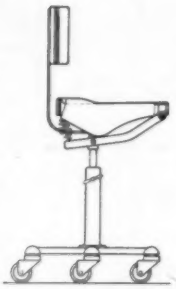

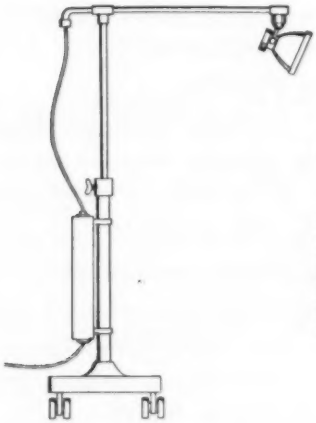

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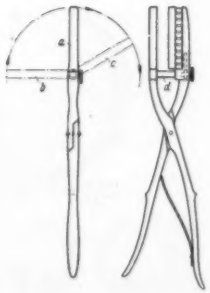

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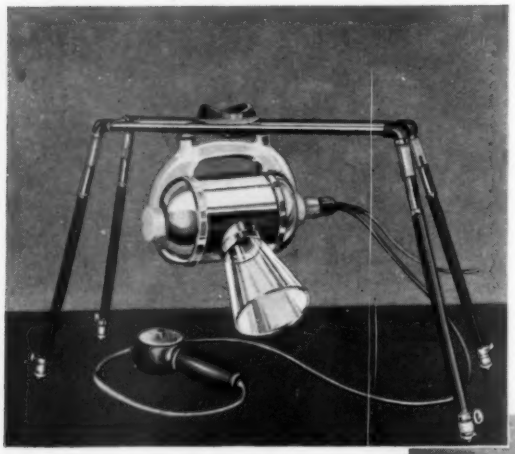

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The Australian and New Zealand Journal of Surgery

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Registered at the General Post Office, Melbourne, for transmission by post as a periodical.

VOL. VI. OCTOBER, 1936. No. 2.

BLOOD VESSEL TUMOURS OF THE SCALP.[1]

By LAMBERT ROGERS,
Honorary Surgeon, Cardiff Royal Infirmary, Professor of Surgery, University of Wales.

IN their masterly account of the tumours which arise from the blood vessels of the brain, Cushing and Bailey[1] distinguished angiomatous malformations from hæmangioblastomata. The same distinction would appear to hold in the case of the blood vessel tumours of the scalp. Known by a variety of names, such as cirsoid aneurysm, plexiform or racemose angioma, aneurysmal varix, varicose nævus, aneurysm by anastomosis, plexiform aneurysm *et cetera*, the majority of these not very uncommon lesions represent developmental anomalies in the vascular bed of a particular region of the scalp rather than true tumours. Occasionally, however, as in the first of the cases here recorded, neoplastic tissue in the form of a hæmangioblastoma is present in addition to the irregular arterio-venous anastomosis which constitutes the bulk of the lesion.

In addition to their interest from the point of view of their pathological classification, these lesions present problems in regard to the most satisfactory method of dealing with them. If untreated, they menace the patient's life from the liability to hæmorrhage, the result of injury or ulceration, while the buzzing, whirring and throbbing which take place in them may so focus the patient's attention and disturb his rest as to cause him much distress. If treated ineffectively, they tend to recur, while radical treatment always presents the important problem of the control of hæmorrhage, which may be truly alarming if effective precautions are not taken to prevent it.

CASE REPORTS.

Case I was associated angiomatous malformation (cirsoid aneurysm) and hæmangioblastoma in a man, aged twenty-one years.

[1] From the Surgical Unit, the Welsh National School of Medicine, the Royal Infirmary, Cardiff.

A cellar-man, aged twenty-one years, was admitted to the Surgical Unit on February 12, 1932, complaining of headache and a lump on the side of the head. This lump was becoming larger, and at times oozed blood and caused him discomfort. The patient stated that he had been told that it was present when he was born, but was then quite small. He remembered that when he was at school it was about the size of a marble. Two months before his admission to hospital

FIGURE I. Case I. Hæmangioblastoma associated with angiomatous malformation. The lesion has been present since birth.

he began to suffer from headaches and attacks of shooting pain in the region of the lump, and he found that it was tender when it was irritated in any way, such as by taking off his cap or brushing his hair.

Examination revealed a dark red scalp tumour, about the size of a plum, lying to the left of the mid-line and just above the frontal hair line. The surface of the tumour was ulcerated and discharged a sero-sanguinous exudate. Around its base, particularly posteriorly and extending well backwards into the left parietal region, was a tortuous cluster of dilated and visibly pulsating vessels (see Figure I). The hair over the bulk of the lesion was normal, but was absent over the dark red tumour. X ray examination of the skull revealed no evidence of increased vascularity in the bone.

Operation was performed on February 15, 1932, under "Novocain" and adrenaline infiltration analgesia. A series of silkworm gut through-and-through sutures was passed through the thickness of the scalp down to the subaponeurotic

FIGURE II. Method of effecting hæmostasis to enable radical extirpation to be undertaken.

space, so that overlapping sutures surrounded the tumour. Inside the line of sutures and around the base of the tumour an incision was made down to the pericranium, and the cut edges of the epicranial aponeurosis were drawn over the

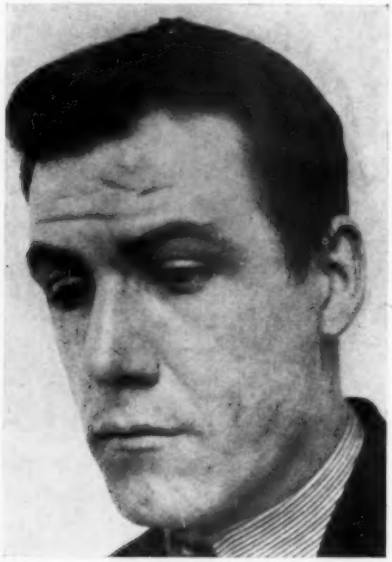

FIGURE III. Case I. The same patient as in Figure I after operation, radical extirpation.

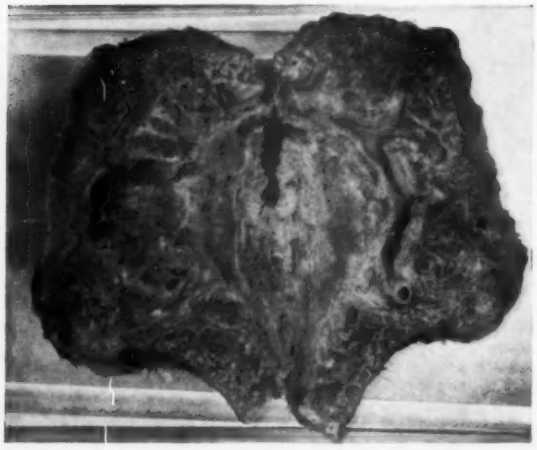

FIGURE IV. Tumour (hæmangioblastoma), Case I.

wound edges in the way that is usually adopted when scalp incisions are made (see Figure II). In this way the lesion was excised, and, as hæmorrhage was effectively arrested, there was very little loss of blood. A number of large feeding vessels were seen to have been severed; cutgut ligatures were placed around these and a dry dressing was applied to the wound.

On February 22, 1932 (one week later), the overlapping silkworm gut sutures were removed. Healing was uninterrupted, and the patient was discharged from hospital three days later. The condition after extirpation is shown in Figure III.

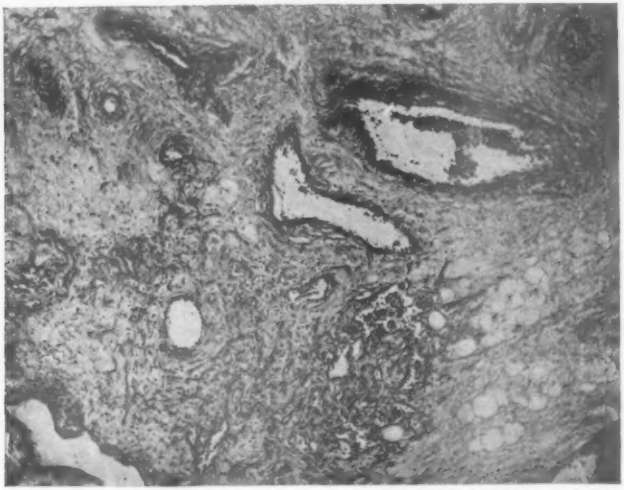

FIGURE V. Microscopic appearance of hæmangioblastoma, Case I.

The patient was seen four years later (February, 1936). The scalp remains cleanly and soundly healed, there is not the least sign of any recurrence of the lesion, and he feels very well. Figures IV and V show the hæmangioblastoma and its microscopic appearance.

Case II was an angiomatous malformation (pure cirsoid aneurysm) in a man, aged twenty years.

A carpenter, aged twenty years, was admitted to the Surgical Unit on January, 20, 1936, complaining of headache, of the presence of a swelling over the back and right sides of his head, and of buzzing, whirring and throbbing noises in the head, especially when he was lying down and trying to go to sleep. Recently these noises had become louder and at times awakened him from sleep.

Examination revealed an extensive, diffuse, pulsating swelling lying over the right side of the skull, extending forwards as far as, and slightly to the right of, the root of the nose and crossing to the left side in the occipital region (see Figure VI). The hair over the swelling was normal. The occipital, superficial temporal and frontal arteries were enlarged, tortuous, distended and pulsating forcibly. X ray examination showed no increased vascularity of the bones of the skull and no evidence of any intracranial lesion.

Operation was performed on January 22 under "Novocain" and adrenaline analgesia. The diffuse pulsatile swelling was all but surrounded by a series of

FIGURE VI. Case II. Man, aged twenty years. Three views of diffuse and extensive lesion (angiomatous malformation only) causing loud noise which distracted the patient.

silkworm gut overlapping sutures, a narrow pedicle of normal scalp being preserved in the left temporal region. A curved incision was then made within the line of overlapping sutures and a scalp flap was reflected, bleeding from the cut edges being arrested by turning over the edge of the epicranial aponeurosis with small artery forceps placed closely together in series (see Figure II). The greater part of the vascular mass was then dissected out, and what remained of the proximal parts of the feeding trunks was ligated with catgut. The flap was resutured in position with interrupted silkworm gut sutures, a small glove drain was inserted at one angle, and a dry dressing was applied. The sutures were released gradually, the last being removed on February 1 (nine days later). The wound edges broke down a little in one or two places, apparently from defective blood supply, but ultimately healing was complete, and Figure VII shows the condition after his discharge.

FIGURE VII. Case II. The same patient as in Figure VI after operation, radical extirpation.

Immediately after the operation he was relieved from his whirring and buzzing noises and headaches. He remains free from this discomfort and is very well.

COMMENTARY.

The Nature of These Lesions.

The two chief forms in which these lesions occur are shown in Figures I and VI. In either case the general appearance of the bulk of the lesion, namely, that

part constituted by the anastomosing vessels, has, with that bizarre conception so often manifest in descriptions of pathological conditions, been compared to that of a pulsating bag of worms.

If we regard an angioma as a tumour composed of an abnormal formation of blood vessels and comprising three varieties, namely, the simple nævus, the cavernous nævus and the plexiform angioma, we admit both the interrelationship of these varieties and the essential similarity

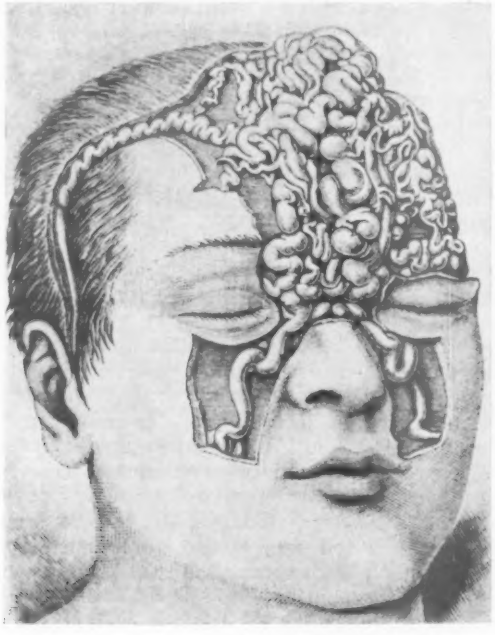

FIGURE VIII. Müller's case, a man, aged twenty years. The lesion was dissected shortly after death.

of the occasionally associated forms such as the facial port wine stain with intracranial angiomatous malformations, an association to which attention has already been drawn.[2]

Sir John Bland Sutton[3] has compared the appearance of the vessels composing cirsoid aneurysms to the normal vascular arrangement of the *rete mirabile* in the forelimb of sloths, the tails of spider monkeys and the subpleural plexuses of cetacens. He refers to an example of the condition recorded by H. Müller[4] in a man, aged twenty years (see Figure VIII). This lesion was first noticed as a red spot when the patient was a year old; at sixteen it began to buzz, and four years later operation

was undertaken by P. Bruns, who ligatured the right and left external carotid arteries and the left common carotid artery; but the patient became hemiplegic on the second day after operation and died on the third. In this case of Müller's the appearance after injection and dissection of the lesion shown in Figure VIII is strikingly like that in the second of the cases here recorded (Figure VI), and both patients were of the same age at the time they were operated upon.

In addition to Müller's case and the two here recorded in detail, additional cases have been reported by R. A. Kerr[5] and A. E. Roche.[6] All these patients were men, and their ages were, respectively, twenty, twenty-one, twenty, twenty-six and twenty-seven years.

Treatment.

In commenting upon the treatment of angiomata in general, Sir John Bland Sutton laid it down that no method is "so safe and effectual as excision, whenever it can be carried out", but stated that "the operative difficulties and dangers in connection with large plexiform angiomas of the head and orbit are very great". Few will disagree with this statement, and for this reason treatment has been suggested by the injection of coagulant fluids, by ligation of the chief vessels (usually the superficial temporal artery or its branches) entering into the formation of the lesion, by the use of electrolysis, diathermy and radiotherapy, and in various other ways. There is no doubt, however, that the ideal treatment is by complete excision of the lesion, since lesser measures are liable to result in recurrence. Excision was therefore practised in both of the cases here recorded. Both patients made satisfactory recoveries, and at no time gave cause for anxiety on account of hæmorrhage, as this was kept in check by the application of two procedures, the first of which was suggested to me by my late colleague, Mr. Robert Cooke, of Bristol. In the first place the lesion was almost surrounded by overlapping sutures of silkworm gut[1] passed through the thickness of the scalp down to the outer table of the skull. These sutures were tied and left in position for a week afterwards. During the excision, hæmostasis was further controlled by reflecting the epicranial aponeurosis (galea) over the edges of the incision, multiple artery forceps, applied closely together, being used for this purpose in the way that is usually adopted in dealing with the scalp when cutting flaps to perform craniotomy. So effective was the combined application of these two procedures that there was practically no blood loss in either of the cases; in fact, in the second case (Figure VII) the hæmostatic sutures were rather too effective, so that some slight sloughing of the wound edges took place later. This would no doubt have been avoided had the pedicle left at one side in the form of a gap between the overlapping sutures been wider (Figure II).

[1] Professor G. Grey Turner tells me that for some time he has used silk for the same purpose.

CONCLUSIONS.

1. The so-called blood vessel tumours of the scalp are developmental anomalies in the vascular bed of the scalp.

2. Two chief varieties occur. They may be either pure angiomatous malformations or angiomatous malformations associated with neoplastic tissue in the form of a hæmangioblastoma.

3. In the above respects they are comparable with the so-called blood vessel tumours of the brain.

4. They are most frequently seen in men between the ages of twenty and thirty years, who complain of headache and of the buzzing and whirring produced by the blood flow through the anastomosing vessels.

5. These lesions are best radically extirpated by dissection, as otherwise (for example, after ligature of feeding trunks only) the bed tends to revascularize and the lesion to recur.

6. The chief problem presented in their treatment is the control of hæmorrhage, and a method is described whereby this can be effected with safety, so that radical excision may be performed.

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RENAL ECHINOCOCCOSIS IN AUSTRALIA AND NEW ZEALAND.

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The advance in urological methods adds a special interest to the study of those cases in which the hexacanth embryo of *Tænia echinococcus granulosis* lodges in the renal cortex, or in which the kidney becomes secondarily infested from preexisting hydatid foci. The condition, though rare, has been responsible for more than 800 reports, no less than 287 in the last decennium. Of the latter, 25, including four personal cases, have originated in Australia and New Zealand. Among the rich material on hydatid disease accumulated by the Registry of the Royal Australasian College of Surgeons are a number of renal cases which illustrate admirably most of the features and vagaries of the disease. These have been generously placed at my disposal by the Registrar-General, Sir Louis Barnett, of Hampden, Otago, and the descriptions have been amplified in many instances by the kindness of the surgeons who originally supplied the reports. It is proposed in this paper to review the twenty-five cases which have already appeared and sixteen from the Registry not hitherto published. The latter will be dealt with in some detail, the others more briefly, as the full reports are available in the original articles. It will be convenient to deal separately with the New Zealand and Australian material, as most of the new cases have occurred in the former country.

Introductory.

While the parasite behaves in the kidney much as elsewhere in the body, there are a few special features which should be borne in mind. The primary cyst is almost invariably in the cortex, but secondary calyx cysts may develop and grow after rupture into the pelvis has occurred. According to their relationship with the pelvis, three types may be distinguished, conveniently designated closed, pseudo-closed and open respectively. In the closed type the cavity enclosed by the pericyst is not in communication with the pelvis; in the second such a communication exists, but the ectocyst remains intact; in the third the ectocyst has ruptured.

The main symptoms correspond to the three groups. In the closed type pressure symptoms alone predominate, in the pseudo-closed type localized intermittent pain with irritation of the urinary tract is added, and in the open type renal colic and the passage of hydatid material

in the urine occur. For descriptive purposes it is useful to use the term "ectocyst" for the whole envelope of the parasite proper, "cyst" for the cavity bounded by the pericyst, and "pericystic" and "endocystic cavity" to denote respectively the space between the pericyst and the ectocyst, and that bounded by the ectocyst itself.

NEW ZEALAND CASES.

CASE I. (Author's case, already published.)

The patient was a female, aged thirty-eight years, single. She was examined in July, 1935. She gave a history of renal colic followed by the passage of cysts on two occasions at an interval of twenty years. The left kidney was removed. Three cysts were present, one in the cortex which had recently ruptured into the middle calyx, and one in the upper and lower major calyces respectively. All the cysts contained numerous daughter cysts. The patient recovered.

CASE II. (Author's case, already published.)

The patient was a male, aged thirty-two years, a clerk. He was examined in January, 1924. He had a hydatid cyst of the left kidney marsupialized at the age of nine. Persistent sinuses resulted. Functionless remains of the right kidney were removed. The patient enjoyed subsequent good health, and had no further evidence of hydatid disease.

CASE III. (Author's case, already published.)

The patient was a female, aged eighteen years. She passed hydatid membrane following right-sided renal colic in the fifth day of the puerperium. She refused investigation or operation.

CASE IV. (From the Royal Australasian College of Surgeons Hydatid Registry, Number 450.) In this case a hydatid cyst was present in the lower pole of the left kidney; nephrectomy was performed, but recurrence took place after six years.

The patient was a female, unmarried. Her age was twenty-three years, and she was a stenographer. She was a town-dweller, and gave no history of contact with dogs. She was examined in March, 1922. Her general condition was good.

She gave a history of having been perfectly well till ten months previously. After that she had a series of mild attacks of pain in the left kidney region. Five months later she had severe left-sided renal colic followed by the passage of "membrane" *per urethram*. Similar attacks followed every three or four weeks, with complete relief in the intervals.

On clinical examination the left kidney was tender and appeared slightly enlarged. The urine was clear, but contained cystic material pathologically verified as hydatid *débris*. No report of X ray examination or cystoscopy was obtained. A diagnosis of hydatid cyst of the left kidney was made. No special tests for hydatid disease were done until after operation (see below).

At operation a cyst was found at the lower pole of the kidney projecting from the anterior surface. The kidney was removed with the greater part of the cyst. The remainder of the cyst wall was curetted and scrubbed with formalin. The pericyst was drained.

The patient was discharged from hospital on the forty-sixth day; the wound was still discharging. The patient had no further trouble for five years, the wound healing shortly after she left hospital. The Ghedini test at that time gave no reaction.

The patient was readmitted to hospital in May, 1928, with a history of recently having passed cysts on several occasions. She had occasional slight

pain on the left side, and suffered from frequency of urination. Scolices were found in the urine. At operation an abdominal incision was made. The right kidney was palpated; nothing abnormal was found. The stump of the left ureter was removed, but showed no trace of hydatid infection. No evidence of recurrence in the left kidney region was noted. The appendix was removed. The wound healed by first intention, and the patient was discharged on the sixteenth day. No symptoms were noted at the end of six months.

Early attacks of mild pain probably indicated a period when the pericystic cavity first communicated with the pelvis, or possibly were due to calyx colic from cysts actually discharged into the lower major calyx. Severe colic denoted either the rupture of the ectocyst or the delayed ureteral passage of cysts lying dormant in the calyx.

The recurrence opens up an interesting problem. There are three possibilities. (i) The first possibility is that daughter cysts were in the ureter at the time of the first operation, and were passed six years later. This can be excluded. Abundant material, including scolices, was passed at the time of recurrence, indicating recent activity. Evidence of this would have been found in the excised ureter. (ii) The second possibility is that there was a small intrarenal cyst in the other kidney not perceptible to palpation at the first operation. This is a possibility, but bilateral cysts, except of the multiple secondary variety, are extremely rare, and the slight pain with the recurrent passage of hydatid elements was on the left side. (iii) The third possibility is that of reformation, from germinal cells of cysts which were left alive on the original pericyst and which reentered the ureteric stump. This is perhaps the most probable explanation. There are no details as to the treatment of the ureter at the first operation, and if the upper end had been in contact with the pericyst, the channel might have remained open, or reopened. As nothing was felt at the second operation, the cyst must have been small. At any rate, the removal of the ureter cleared up the symptoms for the time being. Possibly in another dozen years a cyst will appear subcutaneously in the loin.

CASE V. (From the Royal Australasian College of Surgeons Registry, Number 370, and personal communication.) In this case there was a pseudo-closed cyst at the lower pole of the left kidney.

The patient was a male, aged thirty-eight years. He was examined in February, 1928. He gave a history of having been in contact with dogs *et cetera*; he lived on a sheep farm till the age of twenty-five. His general condition was good. He had an adenoma of the thyreoid removed four years previously. For two years the patient had intermittent attacks of nagging pain in the left loin, but no well-defined renal colic. He had hæmaturia on five occasions in the last year. Following a severe attack, the pain remained as a constant ache in the loin. He never passed membrane or cysts in the urine.

On examination, tenderness was found in the left kidney region. No scolices were found in the urine. No report was received of X ray examination. On cystoscopy no abnormal findings were reported. Function tests revealed the urea in the left kidney urine to be 0·9%; in the right kidney urine it was 1·7%; this indicated diminished functions on the left side. No eosinophilia was found. The diagnosis was "disease of the left kidney".

At operation, the left kidney was explored by the lumbar route. A moderate sized hydatid cyst of the lower pole was found (see Figure I). Nephrectomy was performed, the kidney being removed completely with the cyst.

The patient was well three years later.

The intermittent localized attacks of pain in the kidney region indicated calyx colic. This with recurrent hæmaturia strongly suggests a pseudo-closed cyst.

CASE VI. (From the Royal Australasian College of Surgeons Registry, Number 418.) The condition in this case was a cyst of the upper pole; nephrectomy was performed.

The patient was a male, aged sixteen years. He was examined in March, 1929. He gave a history of contact with dogs *et cetera*; he had lived on a farm.

Early in 1927 the patient had left renal colic and passed gritty material like sand. This was considered to be uric acid sand. In May, 1928, he had another similar attack. The urine then was cloudy and had a trace of albumin. In January, 1929, he had severe pain in the left loin and tenderness along the line of the left ureter. He vomited. No abdominal rigidity was present. In February, 1929, he had another attack and passed a foreign body in the urine; this was identified as laminated membrane. In March, 1929, he was admitted to hospital with the diagnosis of acute appendicitis. The surgeon in the hospital was unacquainted with the details of the previous history. The hospital notes record that for three hours there was severe pain confined to the right side of the abdomen. The pain then eased off. It was fixed and without radiation. No vomiting or nausea occurred.

Case V.

FIGURE I. Hydatid cyst of the lower pole of the kidney.

On clinical examination his temperature was found to be 38·9° C. (102° F.), his pulse rate was 140, and his respiratory rate was 28 in the minute. There was no rigidity. Nothing abnormal was felt in the abdomen. The urine contained

no abnormal constituents. Cystoscopy revealed a normal bladder. Kidney function as shown by the indigo-carmine test was good on both sides. X ray examination revealed no abnormality. Pyelography revealed a normal pelvis on each side. The Ghedini test for hydatid disease gave a positive reaction.

The patient was kept under observation for some weeks. The urine remained normal. Severe continuous pain persisted on the right side of the abdomen. Less severe pain was also present on the left side. A diagnosis of hydatid disease of the urinary tract was made. Its location was undefined.

Exploratory laparotomy was performed to determine the location of the cyst. A normal appendix was removed. A large tumour was felt extending upwards from the upper pole of the left kidney.

A second operation was performed two weeks later. A lumbar incision was made. The kidney and the cyst were removed with difficulty owing to dense adhesions. The cyst was adherent to the hilum of the kidney. Examination of the specimen showed that the upper two-thirds of the kidney had been destroyed. Numerous daughter cysts were present.

Pain on the right side persisted for some weeks, then gradually subsided. At the end of four months all symptoms had disappeared. In the surgeon's opinion the pain was due to inflammation of the diaphragm from pressure of the cyst.

This was an open case. Probably cysts were retained in the pelvis, causing the violent attack for which the patient was admitted to hospital. Crossed renal pain exists in virtue of the inferior mesenteric ganglion being common to both kidneys. The case illustrates the danger of operating without further investigation on the side on which the pain is present. It is difficult to understand how the left pyelogram was normal when the specimen showed two-thirds of the kidney to be destroyed.

CASE VII. (From the Royal Australasian College of Surgeons Registry, Number 357.) This was a case of hilum cyst of the left kidney, with recurrence after eighteen years.

The patient was a male, aged twenty-seven years, a slaughterman. He was examined in March, 1915. He was employed in a freezing works. His general condition was good.

Seven weeks before examination he had severe left renal colic. Two days later he passed a "small white bladder" in the urine. Ever since then he had had pain down the course of the left ureter, with frequency of micturition. Another attack of colic occurred just before his admission to hospital; the pain radiated from the left loin to the tip of the penis. Another "small bladder" was passed two days later.

On clinical examination the kidneys were not palpable. No tumour could be felt. The urine was clear; it contained neither hooks nor scolices. X ray examination revealed no abnormality. An eosinophilia of 8% was present. A diagnosis of hydatid cyst of the left kidney was made.

At operation a lumbar incision was made. The kidney and cyst were removed. There was no record of soiling. The cyst lay in front of the kidney pelvis. Primary suture was used.

The patient had no trouble for eighteen years. Then he developed a fluctuating swelling in the left lumbar region; this was caused by a recurrent cyst. No tenderness and no urinary symptoms were present. He was admitted to hospital for operation in August, 1933. The Ghedini test gave a positive reaction.

At the second operation the cyst was incised, formalinized and stitched, and the wound was closed without drainage. The recurrent parasite extended deeply towards the site of the excised kidney. There were many daughter cysts.

The persistent pain between the first attack and the second indicated the rupture of the ectocyst followed by the retention of daughter cysts in the ureter and pelvis. The case is interesting as indicating recurrence after nephrectomy and the slow growth of the cyst.

CASE VIII. (From the Royal Australasian College of Surgeons Registry, Number 35.) This case is one of septic destruction of the kidney after a cyst of the left kidney had gained communication with the renal pelvis. Uræmia followed.

The patient was a male, aged fifty-two years, a labourer. He was examined in September, 1930. He lived in a country town, but denied any close contact with dogs. He was well nourished, but was in great distress when seen during an attack.

For the last five years he had had, about two or three times a year, severe attacks of left lumbar pain with hæmaturia. After the attacks he passed numerous typical hydatid cysts, seen and diagnosed as such. For the three days before admission to hospital he had suffered from a severe attack with persistent vomiting. Frequency of micturition had followed most of the previous attacks.

He was suffering from great pain on his admission to hospital. His temperature was 38·3° C. (101° F.). There was no visible swelling in the abdomen. An indefinite hard fixed mass was felt below the left costal margin. No fluctuation was present and no friction or thrill. The urine was loaded with pus and granular casts, but no hooklets were found. No X ray or cystoscopy report was received. The renal function was poor. The non-protein nitrogen was 120 milligrammes per 100 cubic centimetres. No special hydatid tests were made.

Vomiting persisted for a month. His general condition became bad, but it improved under treatment, which included the intravenous use of urinary antiseptics. His renal function improved, and the non-protein nitrogen was reduced to 36. It was then considered safe to operate on him. A diagnosis of hydatid cyst of the left kidney with secondary infection was made.

At operation on September 23, 1930, the abdomen was opened by a left paramedial incision. The left kidney was found much enlarged, with dense adhesions to the intestines, colon and spleen. The contour was regular; no individual cyst projected, the whole kidney being involved. The mass was firm. No fluctuation was detected. The right kidney felt normal to palpation. On an attempt being made to separate the left kidney, there were free bleeding and collapse, necessitating the abandonment of the operating. Bleeding was stopped by gauze packing brought out through a stab wound in the loin. The patient was discharged from hospital a month later in fair condition. No report as to his subsequent condition was received.

The case is an example of the rather rare type in which destruction of one kidney and impairment of the other, through sepsis, puts the patient's life in danger. The difficulty of the abdominal route of approach in very adherent cases is illustrated. This difficulty is, of course, not peculiar to the transperitoneal incision.

CASE IX. (From the Royal Australasian College of Surgeons Registry, Number 167.) In this case there occurred an initial attack of renal colic from the left kidney cyst, simulating acute abdominal crisis.

The patient was a male, aged forty-three years, a jeweller. He was examined in July, 1923. He was a town-dweller and kept a dog. His general condition was good.

He had been in good health all his life, his only illness being pleurisy at the age of sixteen. For the last few years he had had occasional slight "indigestion pains". For three weeks before admission into hospital he had been engaged in strenuous physical work—scrub cutting and heavy lifting. After his evening meal he was sitting by the fireside when he experienced sudden and excruciating pain in the left side of the abdomen. He felt sick and vomited. He was examined by his doctor and was sent to the hospital for immediate operation with the diagnosis of "acute abdomen".

Clinical examination six hours after the onset of pain revealed extreme tenderness and rigidity on the left side of the abdomen. Respiration was entirely thoracic. The pain extended particularly along the course of the eleventh dorsal nerve. The result of the examination of urine was not reported. Special hydatid tests were not done till after the operation. No X ray examination was made owing to the supposed urgency of the case. The diagnosis was an acute abdominal crisis, nature undetermined.

Operation was carried out immediately after the patient's admission to hospital. A mid-line abdominal incision was made. The only abnormal finding was a cystic swelling attached to the lower pole of the left kidney. The wound was extended through the left rectus so that better access might be obtained. The cyst was punctured and clear hydatid fluid was withdrawn. On opening the cyst many daughter cysts were evacuated; the cavity was treated with 1% formalin solution. Drains were inserted into the cavity and in the mid-line.

After the operation there was much discharge of urine from the tube in the cavity. The wound was still discharging pus after three months. It was then curetted, and an abscess cavity "the size of an orange" was discovered.

After operation certain investigations were carried out. In the urine blood cells were found, but no hydatid elements were present. X ray examination revealed a shadow in the liver suggesting the presence of a hydatid cyst. Special hydatid tests were carried out. The Ghedini test gave a positive reaction on four occasions, the Casoni test gave no reaction, and an eosinophilia of 2% was present. The sinus was still present at the end of one year.

No gross hydatid elements were passed in the urine or found after operation, thus indicating retention of cysts in the kidney pelvis. The severity and location of the pain suggesting an abdominal crisis, such as perforation of a viscus, was striking.

CASE X. (From the Royal Australasian College of Surgeons Registry, Number 29, and personal communication.) In this case an upper pole cyst caused the first symptoms during pregnancy.

The patient was a female, aged forty-six years, a farmer's wife. She was examined in August, 1930. She had lived on a sheep farm. Her general condition was good.

Till recently she had enjoyed good health. She had three children, aged four years, three years and three months respectively. Her confinements had been normal. A year before her admission to hospital, when she was at the beginning of the last pregnancy, she had for the first time a severe attack of pain in the loin, radiating to the groin and thigh of the right side. Following the attack she passed in the urine a small "round reddish mass". On account of pregnancy no investigation was carried out. She remained well till the seventh month, when she had a similar but not so severe an attack. A third attack occurred during the puerperium after a normal labour. After that she continued to pass at intervals "little bits of skin and bladders, some full and some empty". The pain passed off when the cysts ceased to appear. There was no evidence at any time of anaphylaxis. The last cyst was passed two weeks before her admission to hospital.

Clinical examination showed that the kidneys were not enlarged. The lower pole of the right kidney was palpable, but there was no tenderness or rigidity.

The urine was clear; no hydatid elements were present. Cystoscopy failed to reveal any abnormality in the bladder. The urine from the right kidney contained no hydatid elements. Plain X ray examination revealed no abnormality. In a pyelogram, however, the right kidney shadow showed distortion and flattening of the upper calyces. The renal function was excellent. Her blood non-protein nitrogen figure was 33. In the first hour 30% of injected phenolsulphonephthalein was recovered, and the second hour 45%. The urea concentration test result was 2·4%. Special hydatid tests were carried out. The Ghedini test gave a strongly

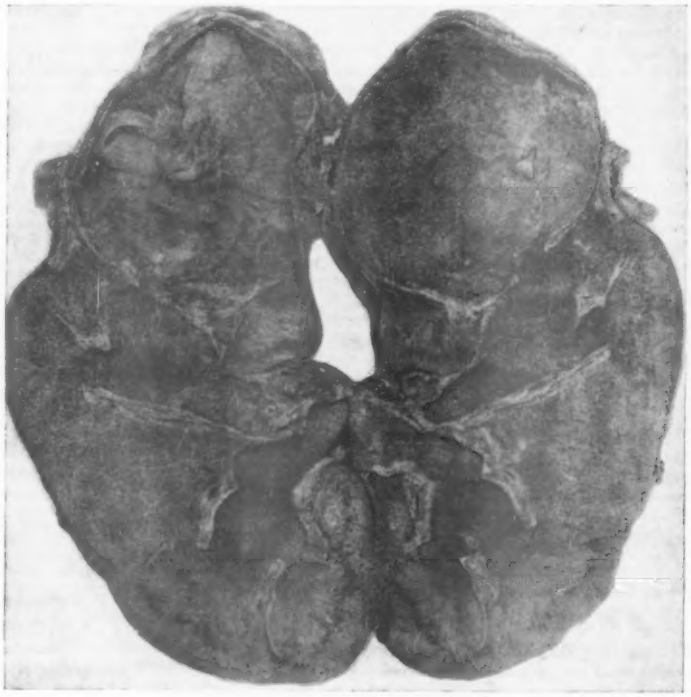

FIGURE II. Hydatid cyst of the upper pole of the kidney. Case X.

positive reaction. The Casoni test gave an immediate positive and a delayed negative result. No eosinophilia was present. The diagnosis was hydatid cyst of the upper pole of the right kidney.

At operation a lumbar incision was made with removal of the twelfth rib. A cyst the size of a billiard ball "sat" on the upper pole of the kidney. The adrenal body was spread over the summit of the cyst and was separated with difficulty. The kidney together with the cyst was removed. The cyst showed no degenerative changes. It contained about a dozen daughter cysts and the collapsed ectocyst (see Figure II). The wound had healed on the twelfth day. The patient was discharged on the twenty-sixth day. She was perfectly well five years later.

The noteworthy points in this case are the commencement of the symptoms during pregnancy and the difficulty in saving the adrenal body.

CASE XI. (From the Royal Australasian College of Surgeons Registry, Number 233.) In this case there was a cyst of the upper pole of the kidney, with a secondary calyx cyst in the lower pole.

The patient was a female, aged thirty-four years, married. She was examined in September, 1933. She had lived for seven years in a country town. Her general condition was good.

For some time she had had occasional attacks of pain in the left loin, followed by the passage of "white grapes" in the urine. For the previous three weeks pain in the loin had been persistent. One week before her admission to hospital she had an attack of hæmaturia, and passed another grape-like body. This proved on pathological examination to consist of laminated hydatid membrane.

Clinical examination revealed a tumour extending 5·0 centimetres (two inches) below the left costal margin. It was smooth and rounded, felt solid, moved with respiration, and could be determined by bimanual examination to be associated with the kidney region. Its internal margin reached the mid-clavicular plane. No report was received regarding a bladder specimen of urine. On X ray examination no abnormal findings were recorded. On cystoscopy the right ureteric orifice was normal; the left was rugged and patulous. The appearance was similar to that seen after the recent passage of stone. Both ureters were easily catheterized, and specimens of urine were obtained. The urine from the right kidney was normal. The urine from the left kidney contained pus cells and hooklets; it yielded no growth on culture. The Ghedini test gave a weakly positive reaction. The Casoni test gave a positive reaction. A diagnosis of hydatid cyst of the left kidney was made.

At operation a paramedial abdominal incision was made. The upper two-thirds of the left kidney were occupied by a hydatid cyst which was densely adherent to the spleen, pancreas and suprarenal gland. After formalinization and tapping, the cyst was opened and evacuated. About half the cyst and the rest of the kidney were removed. A drain was left down to the remaining pericyst and was brought out through a stab incision in the loin. Examination of the specimen showed that the upper two-thirds of the kidney were destroyed by the cyst. On section a smaller cyst was found in the lower pole. The main cyst was degenerate. The internal surface of the ectocyst was like a granulating surface, to which one solitary daughter cyst adhered. The cyst in the lower pole was healthy and contained daughter cysts.

This case approached self-healing through evacuation and infection of the main cyst. The lower cyst was doubtless a secondary one in the lower major calyx derived from a scolex which had escaped at some previous time from the upper cyst.

CASE XII. (From the Royal Australasian College of Surgeons Registry, Number 30, and personal communication.) In this case a septic closed cyst presented as a superficial loin abscess.

The patient was a female, aged sixty-three years, married. She was examined in June, 1930. On inquiry regarding her contact with dogs *et cetera*, it was learned that she lived in town. No report of her early life could be obtained. She was pale and toxic.

Her history revealed no record of symptoms until eight months before her admission to hospital. At that time a painless swelling developed over the right costo-vertebral angle. Three weeks before her admission the swelling became tender and the skin over it red. It was diagnosed as a perinephric abscess. The swelling had gradually increased in size from the time when it

was first noticed. When it was incised thick yellow pus escaped, together with fragments of hydatid membrane and a few small cysts. She had had a strangulated hernia in 1929 and erysipelas in the same year.

On clinical examination the original superficial swelling was soft and fluctuated. After it was drained the acute symptoms subsided and the condition was more completely investigated. There was no history of the patient's passing anything abnormal and no hydatid elements were found in the urine, which was clear. X ray examination revealed a typical curvilinear outline of a calcified hydatid cyst in the right upper part of the abdomen in the kidney region. There was no report regarding cystoscopy. The Ghedini test gave a weakly positive reaction; the Casoni test gave no reaction. No eosinophilia was present. A diagnosis of hydatid cyst of the right kidney or of the liver was made.

At operation in July, 1930, a lumbar incision was made. A cyst with a calcified wall was found. It appeared to be attached both to liver and kidney. The hydatid fluid was purulent. The cyst was formalinized, and cysts and *débris* were removed. The cavity was drained with a tube. The hydatid fluid removed was free from bile and blood. The patient was discharged from hospital three months later with a sinus.

The patient was admitted to hospital again in January, 1933. She had developed urinary symptoms. The urine contained pus. A pyelogram of the right kidney showed hydronephrosis and distortion of the upper major calyx. The diagnosis of non-communicating hydatid cyst of the right kidney was made. The patient refused further operation and left the hospital. In November of the same year she had an apoplectic seizure and died. No autopsy was made.

The history of erysipelas suggests the possibility of anaphylaxis. The subsequent history of pus in the urine and the distortion of the calyx indicate that the cyst was a kidney cyst.

CASE XIII. (From the Royal Australasian College of Surgeons Registry, Number 400.) In this case a pseudo-closed cyst of the left kidney was treated by marsupialization.

The patient was a male, aged forty-eight years, a farmer. He was examined in June, 1927. He gave a history of having worked sheep dogs, and was a fairly well developed man.

He had suffered from diarrhœa for eleven years. He had a dragging pain behind the navel and fullness in the epigastrium after meals for three months. The pain came on immediately on taking food. He had a good appetite, but was afraid to eat. He had shortness of breath on exertion for three months and gradual loss of strength and energy. The pain lasted for two hours. The feeling of fullness, but not the pain, was relieved by alkalis.

Clinical examination showed that the abdomen moved on respiration. There was a fullness of the lower part of the abdomen and the muscles were tense. Tenderness was present on palpation over the umbilicus and half-way between this and the pubis. A tumour was palpable, and there was dulness on percussion from the left costal margin to the umbilicus. There was no record of abnormality in the urine. On cystoscopy the bladder was normal. No report on the renal function was made. No findings on X ray examination were recorded. A pyelogram of the right kidney was normal; a pyelogram of the left showed a filling defect of the lower calyces. The Ghedini test gave no reaction. The Casoni test gave an indefinite reaction. An eosinophilia of 7·5% was present. A diagnosis of splenic enlargement was made.

At operation a lumbar incision was made and the twelfth rib was removed. A cyst was found attached to the lower pole of the left kidney. Clear fluid was withdrawn. Formalin was injected, and the fluid contents were aspirated. Part of the pericyst was excised, the rest was sutured to the wound, and a tube was left in. There were many intact daughter cysts. Slow healing occurred. On one occasion after irrigation with hydrogen peroxide, gas was passed *per*

urethram. The patient was discharged from hospital three months after operation. The patient was seen on several occasions later on; there was no further evidence of hydatid disease.

The communication between the pericystic cavity and the kidney pelvis was demonstrated by the passage of gas after the ectocyst was removed at operation. The diagnosis of enlarged spleen was made, apparently owing to the ambiguous results of the serum and skin tests in spite of the eosinophilia and the positive pyelogram.

CASE XIV. (From the Royal Australasian College of Surgeons Registry, Number 172.) In this case a large left-sided cyst was marsupialized. Death followed empyema and cerebral thrombosis.

The patient was a male, aged forty-six years, a grocer. He was examined in August, 1924. Contact with dogs *et cetera* was not established; he was a town-dweller. He was fairly healthy and well nourished.

Twenty-four years before examination he sustained a severe strain in crossing a flooded stream. He had suffered more or less since then with ill-health. For four years he had had intermittent severe indigestion, with much abdominal pain of a dragging character in the epigastrium, increasing in intensity and then passing off. He was constipated. Vomiting occurred at infrequent intervals. The pain was relieved by bringing up wind. All symptoms had been worse of late. He was sent into hospital with the diagnosis of "recurring appendicitis".

On clinical examination there was a large palpable mass in the right lower part of the abdomen. The contour was fixed, but indefinite. Marked tenderness and rigidity were present to the right of the umbilicus. His temperature was 38·3° C. (101° F.); his pulse rate was 100. Rectal examination revealed no abnormality. The right side of the abdomen was dull to percussion. Nothing abnormal was reported from examination of the urine. There were no reports of cystoscopy and renal function. X ray examination with a bismuth meal showed the stomach raised to close below the liver, with a fixed pylorus suggesting adhesions. Special hydatid tests were not done till after operation. A diagnosis of recurring appendicitis was made.

At operation a high right paramedial incision was made. The caecum and colon were found markedly distended with a fæcal mass. There was "subacute inflammation" in the appendix. Appendicectomy was performed. Further exploration disclosed a cyst larger than an ostrich egg apparently attached to the lower pole of the left kidney. Another incision was made in the left flank, and the cyst was sutured to the skin margins. The cyst was opened. There were "pints" of hydatid material, including daughter cysts from the size of shot to that of a small orange. The cyst was formalinized, cleaned out and two drains were inserted. The hydatid fluid contained no blood, bile or pus. No scolices or hooklets could be detected in the fluid or cysts removed.

Two days after operation the Ghedini test was performed; it yielded no reaction. On the fourteenth day the patient became very ill, with purulent expectoration and high temperature. No hydatid material was found in the sputum. An empyema cavity filled with thick yellow pus and tracking behind the pericardium was opened by removal of ribs in the left axillary region. Later cerebral thrombosis with left-sided paralysis of limbs and aphasia occurred, and death took place three months after operation.

CASE XV. (From the Royal Australasian College of Surgeons Registry, Number 26.) In this case there were primary cysts in the left kidney and suprarenal body. The primary cysts ruptured into the peritoneal cavity.

The patient was a female, aged fifty-two years, married. She was examined in May, 1930. Contact with dogs *et cetera* could not be determined, for there was no early history; she was a town-dweller. She was extremely ill.

Her history was not ascertainable, except that she had had dyspnœa for six weeks with loss of appetite and gradual onset of delirious attacks. She suffered from cough, with blood-stained sputum and anorexia.

Clinical examination revealed extreme general œdema. She looked old and very weak, being propped up in bed. She had difficulty in speaking. Her pulse rate was 140. The apex beat was 5·0 centimetres (two inches) outside the nipple line. There was a mid-line abdominal scar. The results of urine examination *et cetera* were not reported. The diagnosis was cardiac failure with œdema.

On the seven day after her admission to hospital the patient developed axillary vein thrombosis, and died on the ninth day.

Autopsy revealed a gangrenous ulcer on the left arm below the elbow. In the abdomen there were large numbers of hydatid cysts in the mesentery, peritoneal coat of the intestine and in the pelvis. Matted cysts in the pelvis were obstructing the left ureter. There was a cyst on the anterior aspect of the right lobe of the liver. One hydatid cyst was found in the left suprarenal gland. The thyreoid was enlarged and nodular. The right pleural cavity was obliterated by adhesions; the left contained free fluid. The lungs contained infarcts, but no hydatid cysts. The left kidney was entirely disorganized by one large hydatid cyst. The right kidney contained no cysts; it was hypertrophied and hydronephrotic.

The reporter of the case was of the opinion that a cyst of the left kidney had ruptured into the peritoneal cavity, the small liver cyst being a secondary.

CASE XVI. (From the Royal Australasian College of Surgeons Registry, Number 533.) In this case a huge open cyst of the right kidney was treated by marsupialization.

The patient was a male, aged about forty years, a shepherd. He was examined in 1908. (Note: This is an old case recently sent to the Registry.) He gave a history of having worked sheep dogs. He looked rather worried and pulled down.

For many months he had suffered from a dull ache over the right loin, with frequent severe attacks of colic, after which he passed blood and "skins" in the urine. His chief complaint was pain and distress in the bladder owing to the inability to pass the cysts through the urethra. He had had attacks of complete retention of urine. He had no symptoms apart from the urinary system. Attacks of hæmaturia accompanied the passage of cysts.

On clinical examination there was great bulging in the front and back over the right loin. The swelling was very tense, fixed and dull to percussion. No hydatid thrill was detected. The urine contained blood, albumin, pieces of membrane and daughter cysts. X ray examination was not available.

At operation a loin incision was made. The cyst was opened and its contents were emptied. There was "half a bucketful" of daughter cysts of all sizes. The cyst was marsupialized and drained. Recovery was uneventful. The patient resumed active work. There was no late "follow-up".

It is probable from the report that the whole kidney had been destroyed and replaced by the cyst.

CASE XVII. (Personal communication. Now on Royal Australasian College of Surgeons Registry, Number 562.) This was a case of general echinococcosis with bilateral hydatid disease of the kidneys.

The patient was a female, aged about sixty years, a Maori. She was examined in 1932. She had lived in the crowded conditions of a Maori settlement in the country. She was extremely ill. Her history was not obtainable.

She was emaciated to a skeleton, except for an enormous protruding abdomen. She was very drowsy and showed other symptoms of uræmia. No

report was made about the urine. No diagnosis was made. The patient gradually sank and died on the fourteenth day after her admission to hospital.

At autopsy the whole abdomen was filled with innumerable hydatid cysts, with daughter cysts lying loose in the peritoneal cavity. The right kidney was so studded with hydatid cysts that it resembled a polycystic kidney. The larger of these cysts contained daughter cysts. The left kidney was similar to the right, but the cysts were not so numerous. The liver contained several large cysts.

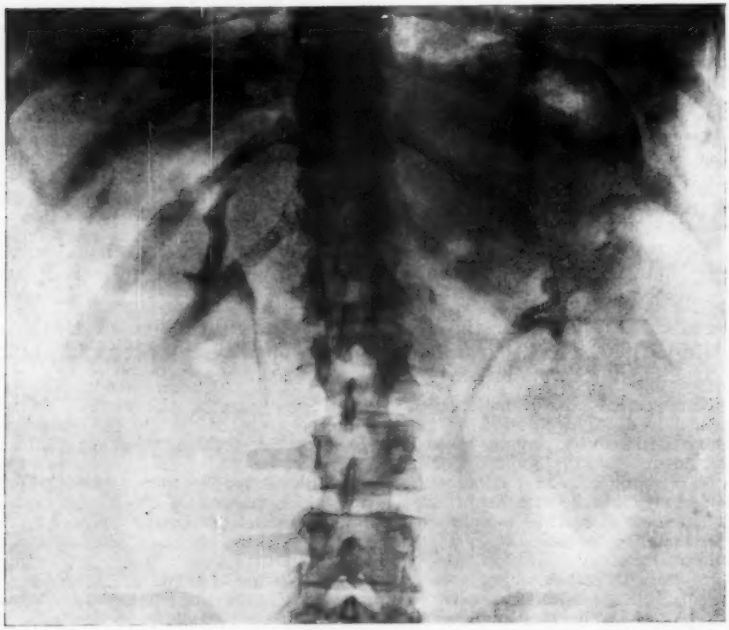

FIGURE III. Pyelogram (intravenous) of the kidney in Case XVIII. The spherical calcified hydatid is seen in apposition with the upper calyx and overlapping the upper pole. The lower part of the kidney and the remainder of the pelvis are normal.

The presence of free daughter cysts indicated the recent rupture of a cyst, either in the liver or elsewhere in the abdomen. The rupture from the liver into the peritoneum had also taken place at some previous time, as well as the embolic spread to the kidneys, as witnessed by the presence of daughter cysts in the latter.

CASE XVIII. (Reported to the Auckland Clinical Society. Roche and Mackenzie's case.)

The patient was a female, aged forty-seven years. She was examined in December, 1934. For eighteen months she had had slight intermittent pain in the lower thoracic region on the left side. She had no urinary symptoms; the urine was normal. An intravenous pyelogram showed downward displace-

ment of the left kidney with partial obliteration of the upper calyces (see Figure III). A large rounded mass with a calcified border was seen, continuous with the upper part of the renal shadow. The mass moved freely with respiration. The Ghedini test gave no reaction. The Casoni test gave an immediate negative and a delayed strongly positive response.

At operation a lumbar incision was made with removal of the twelfth and eleventh ribs. A calcified cyst nine centimetres in diameter was found attached

FIGURE IV. Plain skiagram of the kidney in Case XIX. The two intersecting curvilinear shadows are visible.

to the upper pole of the left kidney. Sudden collapse occurred, and the patient died on the third day. There was no autopsy.

CASE XIX. (Author's case, already published.)

The patient was a male, aged fifty-three years. He was seen in consultation in November, 1935. A routine X ray examination for suspected neoplasm of the lung revealed two intersecting ring shadows typical of hydatid cysts in the

FIGURE V. Posterior view of the kidney in Case XIX.

neighbourhood of the left kidney (see Figure IV). The Casoni and Ghedini tests gave positive reactions. No eosinophilia was present. The patient died of his lung trouble. At autopsy there were two hydatid cysts in the left kidney, and none elsewhere in the body (see Figures V, VI, VII, VIII and IX). The diagnosis of malignant neoplasm of the lung was confirmed. The pericystic cavity of the two cysts communicated with each other and with the middle major calyx. The ectocysts were, however, intact.

REMARKS ON THE NEW ZEALAND CASES.

It will be of interest to draw attention to the outstanding points of the nineteen cases just recorded. They present an excellent cross-section of the subject, and one or two of them are of unusual interest.

Incidence.

Barnett estimates that more than a hundred cases of hydatid disease occur yearly in New Zealand. As eighteen of the kidney cases occurred within the last dozen years, the percentage is about 1·5. Most of the cases have, no doubt, found their way to the Registry. Ten of the nineteen occurred in the North Island, nine in the South. Otago and Wellington provinces furnished the majority.

Pathological Features.

In all cases but one (Case XVII) the renal cysts were primary; in four they were multiple. Case XIX is of

FIGURE VI. Anterior view of the kidney in Case XIX. The two separate cysts are clearly seen. Windows have been cut in their anterior walls.

particular interest. It is very rare to find a double primary renal infestation, but in this instance a complete autopsy definitely excluded cysts elsewhere in the body. Cases I and XI represent a different type—a primary cortical cyst with secondary implantations in the calices through rupture at some previous time. In Case XVII both kidneys were full of cysts arising by embolic spread. This was the only instance in which the disease was bilateral, unless it is accepted that the recurrence in Case IV was due to an undiscovered focus in the remaining kidney.

FIGURE VII. Skiagram of the excised kidney in Case XIX, anterior view.

FIGURE VIII. Skiagram of the excised kidney in Case XIX, lateral view. The probe lies in the ureter.

Cysts Elsewhere in the Body.

In Case IX the X ray examination disclosed a suspicious shadow in the liver. In Case XV there was an associated cyst in the left suprarenal gland which was in all probability a primary one; the other numerous hydatids scattered about the abdomen were due to rupture of the enormous kidney cyst into the peritoneal cavity—an exceedingly rare event. In no other case was there evidence of other cysts in the body.

Sex and Age Incidence.

There were nine female and ten male patients. The youngest patient was five, the oldest sixty-three years of age. The heaviest incidence

was in the fourth and fifth decades, as seen in the following tabulation:

First decade (birth to 10 years)	1
Second decade (10 to 20 years)	2
Third decade (20 to 30 years)	2
Fourth decade (30 to 40 years)	5
Fifth decade (40 to 50 years)	5
Sixth decade (50 to 60 years)	2
Seventh decade (60 to 70 years)	2
Total	19

The age period stated is the age of the patient at the time when medical advice was first sought; it in no way represents the period of infestation, which, as Dew has pointed out, is as a rule in childhood.

Side Affected and Site of Implantation.

The side affected and site of implantation were as follows: left kidney, 15 cases; right kidney, 3 cases; bilateral, 1 case.

The preponderance of left-sided cases is remarkable, as in large series of cases the kidneys are affected with equal frequency (Nicaise).

Position of Cysts.

The position of the cysts was as follows: upper pole, 5 cases; lower pole, 7 cases; upper two-thirds, 1 case; hilum, 1 case; convex border, 1 case; whole kidney, 2 cases; multiple secondary, 1 case; unknown, 1 case; total, 19.

Other Pathological Features of Interest.

Case I was remarkable for the com-

FIGURE IX. Pyelogram made on the excised specimen in Case XIX. The cyst was of the pseudo-closed type, but the pressure of the opaque medium has ruptured the ectocyst and penetrated into the endocystic cavity, outlining daughter cysts.

paratively insignificant damage to the kidney and lack of symptoms, in spite of three active cysts. Case II gave an insight into the possible after-history of marsupialization. This unfortunate patient had been condemned for twenty-three years to running sinuses in the loin and abdomen. Sepsis had destroyed the kidney, but fortunately also occluded the ureter, so that he was spared urinary discomfort. The first symptoms of trouble frequently date from a pregnancy or from trauma. Cases III and X were instances of the former, Case XIV of the latter. The stress of labour pains may convert a pseudo-closed cyst into an open one. Case IV is of extraordinary interest and I think unique. It shows how a ureter may act as the excretory organ for the contents of a hydatid cyst, even when the kidney is removed, and yet remain itself uninfected, probably in virtue of the maintenance of its peristaltic action. In Case I and in many cases in the literature cysts were actually present in the ureter at the time of operation, and yet no case of secondary ureteric cyst has occurred. There is accordingly no need to perform complete ureterectomy in cases of renal echinococcosis. Case VI illustrates a common mistake in diagnosis. There was an open cyst in a boy of fourteen, and, as in most young subjects, it was univesicular. The only hydatid material passed was "sand"; this was mistaken for ordinary uric acid gravel, and not examined microscopically. The diagnosis was accordingly delayed for two years until the trauma of rupture had induced the formation of daughter cysts which were finally passed and identified.

Case VII illustrates the very slow growth of a recurrence in the scar after nephrectomy and the comparative innocuousness of the secondary cyst. It more than justified the treatment by primary suture as against marsupialization, even though the former carries perhaps a greater risk of recurrence.

Case VIII illustrates a rare example of echinococcosis of the kidney threatening the life of the patient, the sepsis being conveyed to the other kidney and producing uræmic symptoms. For the most part renal hydatid disease is benign, ample kidney substance being preserved not only in the other kidney but also in the one affected. In Case IX the severity of the renal colic due to rupture of the cyst was no doubt accentuated by anaphylactic phenomena, and the rigidity of the abdominal muscles prevented the discovery of what proved to be a very large cyst. It is probable, however, that a urinary examination would have thrown doubt on the diagnosis of an acute abdominal crisis.

The first sign in Case XII was the presenting of an inflamed swelling in the loin, diagnosed as perinephric abscess. This is a most unusual type, as a hydatid cyst of the kidney rarely (never, according to Craig and Lee-Brown) becomes septic unless it has ruptured into the kidney pelvis. In this instance, however, there was a complication in that the cyst had invaded the liver and may have gained communication with the biliary system, though the cystic fluid was not bile-stained. Few of the cases showed any complications of a serious nature. In Case XIV the fatal empyema may have been due to a spread of sepsis

from the cyst in the left kidney. In Case XV secondary cysts in the peritoneal cavity had blocked the ureter of the uninfected kidney, causing uræmia and death, and in Case XVII general embolic echinococcosis from a cyst unconnected with the kidney produced the fatal result. In Case XVIII the sudden post-operative death was probably due to embolism.

Symptoms.

A careful study of the case histories will reveal in many of them the transition from the mild calyx colic of the pseudo-closed type to the fulminating colic when the ectocyst finally ruptured. In Case XIII the cyst caused pressure symptoms only, but the removal of the ectocyst at operation showed a free communication between the pericystic cavity and the kidney pelvis. The first symptoms may be summarized as follows:

Mild intermittent pain in the kidney region	4 cases
Severe renal colic with passage of hydatid material ..	8 cases
Catastrophic pain resembling abdominal crisis	1 case
Chronic gastro-intestinal symptoms	2 cases
Uræmic symptoms with fatal result	2 cases
Abscess pointing in the loin	1 case
No symptoms	1 case
Total	19 cases

Diagnosis.

The open cases, nine in number, were all ultimately diagnosed before operation through the identification of the hydatid nature of the material passed in the urine. In one a laparotomy was done to determine the side affected, and because of some confusing symptoms leading to the suspicion that appendicitis was present as a complicating factor. This was Case VI. In the light of the early left renal colic, however, the necessity for this procedure is not apparent. Two of the closed or pseudo-closed cysts were diagnosed in both instances through the discovery of a typical ring shadow on routine X ray examination. These were Cases XVIII and XIX. This is a most valuable sign, though not an infallible one. Lee-Brown illustrates a typical ring shadow over the right kidney region and a distorted pelvis in the pyelogram. The cyst proved to be a liver cyst pressing heavily on the kidney. Kretschmer and Moody found a similar ring shadow in a case of adeno-carcinoma of the kidney. Revesz had his diagnosis of renal hydatid disease confirmed by a whole congress of German radiologists on the basis of three exactly circular and overlapping shadows. Illyes, who agreed with the diagnosis, found three smooth spherical calculi at operation! The finding of a ring shadow is, however, most valuable, and, if accompanied by positive reaction to the Ghedini or Casoni test, is sure to indicate a hydatid cyst. Retrograde and intravenous pyelograms should serve to distinguish liver and spleen cysts in most cases, though in Case XIII the error was made. Eight of the cases were not diagnosed until operation or autopsy. In Case V the kidney was explored

for an undetermined pathological condition. The Casoni test would in all probability have led to a pre-operative diagnosis. In Case IX a urinary examination would have thrown doubt on the diagnosis of acute abdominal crisis, and would have prevented an immediate abdominal operation. When the attack had subsided, the large cyst would have been palpable and the diagnosis of hydatid kidney ultimately made. In Case XII the cyst presented as an abscess ripe for opening and cysts and membrane escaped with the pus. Had the patient been seen earlier, diagnosis by X ray examination would have undoubtedly been made. In Case XIV the distension of the colon and the tenseness of the abdomen concealed the large cyst. Had the recently discovered non-irritating pyelographic media such as "Abrodil" been available, the patient would undoubtedly have been subjected to X ray examination and pyelography. Serum tests would then have been performed and a diagnosis made. Large tumours of the kidney, however, may be very difficult to feel. In Case XIX, though we knew that there were two cysts either of the kidney or spleen, repeated palpation of a soft abdomen and with the fingers inserted well under the costal margin failed, except on one occasion, to disclose the tumour. The patient's respiration was quick and shallow owing to the chest condition, and the excursion of the diaphragm was too small to push the kidney down to the examining fingers.

In Cases XV and XVII the patients were in a moribund state when seen; no proper history could be obtained and no examination could be made. These were really the only two cases in which it was theoretically impossible to make a diagnosis.

The literature contains adequate information on the use, value and defects of the complement fixation tests of Ghedini or Weinberg and the intradermal test of Casoni. In the nineteen cases the results of these tests and the blood count for eosinophilia were as follows:

Eosinophile Count: Not reported, 13; 2% or less, 4; eosinophilia, 2 (8% and 7·5%); total, 19.

Ghedini Test: Not done, 9; no reaction, 4; weakly positive reaction, 2; positive reaction, 3; strongly positive reaction, 1; total, 19.

Casoni Test: Not done, 12; no reaction, 2; indefinite reaction, 1; positive reaction, 2; immediate positive, delayed negative reaction, 1; immediate negative, delayed positive reaction, 1; total, 19.

The present position of diagnosis of renal hydatid disease appears to be that all cases in which membrane is passed are diagnosed, but when only "hydatid sand" appears in the urine a microscopic examination is sometimes omitted. If examination of urinary sediment is made as a routine all such cases should be diagnosed.

Obscure cases without palpable tumour which show a round calcified area should, with the help of the serum tests and pyelograms, always be diagnosed.

The same applies to those instances in which a palpable tumour of unknown pathology is felt. When the tumour is in the hypochondrium,

pyelograms will as a rule be obtained; but when the tumour is apparently in the abdomen, far too often exploratory laparotomy is carried out without waiting for the valuable results of pyelograms which, by distortion of the ureters, will at least show whether the growth is intraperitoneal or extraperitoneal.

When closed cysts exist without palpable tumour or visible X ray shadow, only in clinics where very exhaustive routine examinations are carried out will pathological change in the kidney be discovered, and this may lead to the specific serum tests. In most cases the condition will escape diagnosis. There is one other type of upper pole cyst in which the only sign is dulness extending up from one or other lung base. In the absence of chest disease the sign indicates in most cases a massive hydronephrosis or a hydatid cyst of the upper pole.

Prognosis.

Prognosis is good in most cases of kidney hydatid disease. Many escape observation. In Case XIX the calcified cysts of the left kidney did not incommode the patient in the slightest. They were a purely accidental finding. Only three patients died as the direct result of the hydatid disease, one as the sequel of an operation for the relief of symptoms.

Treatment.

The cases illustrate the good results of nephrectomy with removal of the whole of the cyst or as much as possible, together with primary closure. There were eight operations with no death. Recurrence may be taking place in many, but, as in Case VII, the recurrence takes a long time to appear and is relatively innocuous. It is important that the ureter should be cut down well to prevent the recurrent cyst opening into the urinary tract through it, as probably happened in Case IV. The transperitoneal route is unsatisfactory. Direct access is essential, so that the surgeon may be able to deal by sharp dissection with firm adhesions; blind enucleation may lead to trouble or even disaster. Of the four conservative operations carried out by the lumbar route, one, in Case XII, was compulsory (radical operation being impossible), but ended in rapid recurrence; one resulted in a post-operative death; and the others, in which drainage was used, led to very prolonged suppuration and slow convalescence. In certain dormant cases it is doubtful whether any operation is justifiable or necessary.

THE AUSTRALIAN CASES.

Twenty cases of renal hydatid disease occurring in Australia have been published in the last ten years. Two more have been reported to the Royal Australasian College of Surgeons Hydatid Registry. It is proposed to give the latter somewhat fully and to summarize the others.

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CASE XX. (From the Royal Australasian College of Surgeons Registry, Number 512.) In this case a calculus was associated with a hydatid cyst.

The patient was a male, aged thirty-one years, a taxi-driver. He was examined in July, 1935. He had worked among sheep as a boy.

Two and a half years prior to being seen he had an attack of right renal colic and passed "pieces of jelly". Attacks of a similar nature recurred about every three months. Six months before his admission to hospital he suffered from colic and hæmaturia and passed a small stone. From that time he had had a dull constant ache in the right loin and persistent hæmaturia without clots.

On clinical examination no tumour could be palpated. The urine was clear at the time of examination. On cystoscopy no efflux was seen from the right kidney. In the test for renal function, indigo-carmine appeared from the left side in good density in five minutes; there was none from the right. On X ray examination a shadow suggesting calculus was seen over the kidney area. In a pyelogram the pelvis appeared normal; the calculus shadow lay over the kidney pelvis. No special tests for hydatid disease were done. A diagnosis of stone in the right kidney pelvis was made.

At operation the patient was anæsthetized with ether given by the intrapharyngeal method. A cyst 7·5 centimetres (three inches) in diameter was found at the lower pole. The surgeon hoped to treat the case conservatively and, after freeing the kidney, he opened the pelvis and extracted the calculus with forceps. It was then noticed that a small daughter cyst was caught between the forceps and the stone. The cyst and kidney were then removed. A section of the calculus showed that there was a nucleus of hyaline material, which was considered to be laminated membrane. The patient was discharged from hospital on the sixteenth day.

While gall-stones are apparently not uncommonly associated with hydatid cysts that have ruptured into the gall-bladder, the literature of renal cysts contains very few examples of the associated pathological conditions, not more than a dozen in all. Case XXXVII, reported by Craig and Lee-Brown (see below), was somewhat similar to this case, as an oval stone was impacted in the ureter just below the pelvis. After manipulation with a ureteric catheter the stone was passed and followed by a mass of laminated membrane. Needless to say, the diagnosis of hydatid disease is not made in these cases unless there is a palpable tumour or a ring shadow, and seldom even then.

CASE XXI. (From the Royal Australasian College of Surgeons Registry, No. 568.)

The patient was a male, aged sixty-one years. He was admitted to hospital in March, 1934. He was moribund on admission.

Inquiry into the history failed to reveal association with dogs. The patient had been operated on for hydatid disease of the liver twenty years before. Since then he had had vomiting attacks at intervals. He had a nervous breakdown one year before admission to hospital.

On clinical examination the patient was very wasted and extremely ill. His temperature was 38·3° C. (101° F.), and his pulse rate was 126 in the minute. There was some tenderness in the left loin. Symptoms of uræmia were marked. A diagnosis of hydatid cyst of the kidney and of uræmia was made. The patient died on the third day.

Autopsy revealed a hydatid cyst 7·5 centimetres (three inches) in diameter in the upper pole of the right kidney. The cortical definition in both kidneys

was poor. There was a "lump" 2·5 centimetres (one inch) in diameter hanging from the liver in the region of the falciform ligament.

The kidney cyst was in all probability a primary one. There is no reason to believe that it was a contributory factor to the death of the patient. The operation on the liver cyst had apparently resulted in cure.

CASE XXII. (Craig and Lee-Brown.)

The patient was a female, aged twenty-one years. There was a pseudo-closed cyst of the right kidney with urinary symptoms, but no hydatid elements were passed. A large tumour was palpated in the right hypochondrium. The cyst was marsupialized. The patient recovered.

CASE XXIII. (Craig and Lee-Brown.)

The patient was a male, aged forty-three years. A closed cyst filled the left half of the abdomen, pushing up the left diaphragm and causing intestinal obstruction. Marsupialization was carried out with subsequent left nephrostomy. Recovery followed.

CASE XXIV. (Craig and Lee-Brown.)

The patient was a male, aged twenty-six years. An open cyst of the left kidney was present. Nephrectomy was performed and recovery followed.

CASE XXV. (Craig and Lee-Brown.)

The patient was a female, aged thirty-three years. A pseudo-closed cyst of the left kidney was present with bilateral pyelonephritis. The patient died.

CASE XXVI. (Craig and Lee-Brown.)

The patient was a female, aged nineteen years. The patient had had an operation for a hydatid cyst of the liver at the age of seven. A hydatid cyst of the right kidney with painful tumour of the right hypochondrium grew rapidly after pregnancy and labour. Marsupialization was carried out and recovery followed.

CASE XXVII. (Craig and Lee-Brown.)

The patient was a male, aged sixty-seven years. He had a calcified hydatid cyst of the right kidney, causing hæmaturia and an attack of "cystitis". Apparently it was of the pseudo-closed type, as no hydatid elements appeared in the urine. The symptoms abated. No operation was performed.

CASE XXVIII. (Craig and Lee-Brown.)

The patient was a male, aged fifty-eight years. A hydatid cyst of the left kidney presented as a large abdominal tumour with symptoms of calyx colic and frequency of micturition. No membrane was passed *per urethram*. There was an associated large liver cyst. Both cysts were evacuated and closed. Primary suture was used and recovery followed.

CASE XXIX. (Craig and Lee-Brown.)

The patient was a male, aged nine years. He had a symptomless tumour in the right hypochondrium, which proved to be a hydatid cyst of the right kidney. The cyst was marsupialized and recovery followed.

CASE XXX. (Craig and Lee-Brown.)

The patient was a male, aged thirty-eight years. He had a septic hydatid cyst of the left kidney. Hooklets were present in the urine. The cyst was marsupialized. Sudden death occurred two years after operation.

CASE XXXI. (Craig and Lee-Brown.)

The patient was a female, aged fifty-nine years. A cyst occupied the whole of the right kidney and communicated with a bilocular cyst of the liver.

A piece of hydatid membrane was found in the eye of a ureteral catheter. The cyst was marsupialized. Death occurred one year later from rupture of a liver cyst into the pleural cavity and bronchi.

CASE XXXII. (Craig and Lee-Brown.)

In this case a large cyst of the left kidney presented as an abdominal tumour, with frequency of micturition and intermittent colic, but no passage of hydatid material in the urine. The cyst was formalinized and sewn up and anchored to the abdominal muscles. Recovery followed.

CASE XXXIII. (Craig and Lee-Brown.)

The patient was a male, aged fifty years. He underwent nephrectomy for a cyst of the lower pole of the left kidney.

CASE XXXIV. (Craig and Lee-Brown.)

The patient was a male, aged thirty years. He had a cyst of the left kidney with right-sided colic at intervals for twelve years. Nephrectomy was performed and recovery followed.

The crossed renal pain in this case is a warning against making a diagnosis without urological investigation to determine the side affected. Somewhat similar conditions prevailed in Case VI.

CASE XXXV. (Craig and Lee-Brown.)

The patient was a male, aged forty-one years. He had a calcified hydatid cyst of the right kidney with hæmaturia. A diagnosis was made by finding ring shadows in the X ray film. No operation was performed.

CASE XXXVI. (Craig and Lee-Brown.)

The patient was a female, aged sixty years. She had a calcified hydatid cyst of the right kidney with attacks of colic and the passage of "gravel" at intervals for twenty years. The "gravel" proved to be calcified *débris* from the cyst. Nephrectomy was performed and recovery followed.

CASE XXXVII. (Craig and Lee-Brown.)

The patient was a male, aged forty-five years. In this case there was an intrarenal cyst of the left kidney which was so small that it could not be detected till the excised kidney was cut open. An associated calculus was impacted in the upper part of the left ureter.

CASE XXXVIII. (Lee-Brown's case.) No details are given of this case.

CASE XXXIX. (Lee-Brown's case.) No details are given of this case.

CASE XL. (Flynn's case.)

The patient was a female, aged twenty-one years. She had a univesicular cyst of the right kidney which presented as a painless swelling in the right hypochondrium and which increased in size during the puerperium. The cyst contents were evacuated and primary suture was used. Recovery followed.

CASE XLI. (Hurley's case.)

The patient was a male, aged fifty-two years. He had a calcified cyst of the upper pole of the left kidney. From the age of eighteen he had suffered from intermittent attacks of renal colic with the passage of membrane *per urethram*. The cyst contents were evacuated and the remaining cavity was allowed to drain through a communication existing with the upper major calyx. Recovery followed.

SUMMARY.

The paper reviews forty-one cases of hydatid disease of the kidney occurring in Australia and New Zealand mostly in the last decennium. These cases include four personal cases and sixteen hitherto unpublished. Most of the latter are recorded in full in the Registry of Hydatids established by the Royal Australasian College of Surgeons.

An analysis is made of the New Zealand cases.

ACKNOWLEDGEMENTS.

I have to express my sincere thanks to Sir Louis Barnett, who not only gave permission for the publication of the Registry cases, but took a great deal of trouble to secure exact data in regard to them. He also made many valuable comments and supplied much information on the general aspects of hydatid disease which was invaluable for comparative purposes. I have also to thank Professor Gordon Bell, Mr. Stanley Foster, Mr. Gordon Anderson, Dr. Kronfelt and Dr. Putnam for information in regard to some of the case histories. I have to thank Dr. Bruce Mackenzie for the pyelogram in Figure III, and Professor D'Ath, of Dunedin, who kindly provided me with the photographs illustrating Cases V and X.

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TUMOURS OF BONE.

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Before an attempt is made to describe the different tumours of bone, certain facts should be borne in mind. Bone is merely a type of connective tissue, in which the ground substance is impregnated with salts of lime, so that certain tumours of bone will resemble tumours

Figure I. Areas of rarefaction in the upper extremities of humeri and scapulæ.[1]

found in connective tissue elsewhere. The fibromata and fibrosarcomata of bone resemble similar tumours occurring more frequently in the subcutaneous tissue, fascia and muscle, so that the presence of this kind of tumour in bone is merely fortuitous. The only tumours which can be regarded as being peculiar to bone are those originating in the osteoblast and osteoclast and their precursors, namely the osteomata, osteogenic sarcomata and the giant cell tumours. The origin of certain other tumours of bone is still obscure. The malignant tumour associated with the name of Ewing was believed by him to originate in the perivascular lymphatic endothelium, and was described by him as an endothelial myeloma. Others consider that it arises from the reticulo-endothelium of the marrow, and they describe it as a reticulum cell sarcoma.

[1] Figures I, II and III were considered by the radiographer to be typical of lipoid histiocytosis. *Post mortem* examination did not reveal any lipoid cells, and the condition was reported by the pathologist as being osteogenic sarcoma. Numerous secondary nodules were found in the lungs.

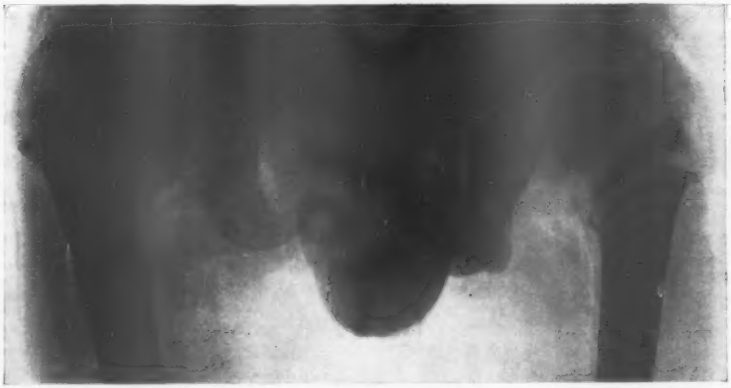

FIGURE II. Areas of rarefaction in the upper extremities of the femora and pelvic bones.

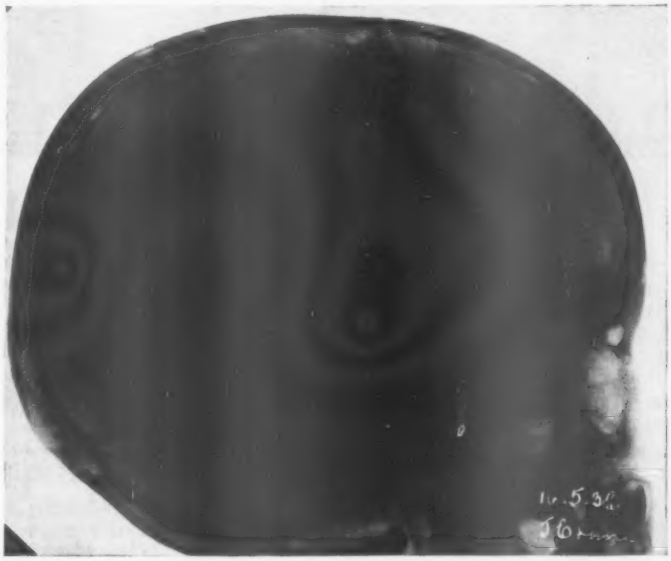

FIGURE III. Areas of rarefaction in the calvarium (mapping of the skull).

Other common types of bone tumour, such as the multiple myelomata, are regarded by some authorities as a disease of the blood-forming organs rather than true tumours. Multiple myelomata, however, seem to occur only in late adult life, and I can find no instance of this condition in young children. In early life there occurs, although with great rarity, a similar condition, namely, chloroma, and this disease, like multiple myelomata, forms a link between leuchæmia and tumour growth, as it is a malignant tumour with a leuchæmic blood picture. In this disease, green tumours are found in the group of bones which are commonly affected in multiple myelomata, namely, the flat bones, such as the sternum, ribs, pelvic bones and bones of the skull.

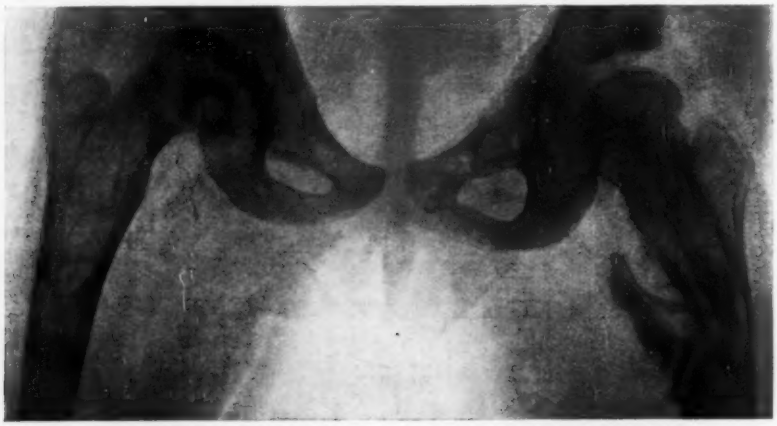

FIGURE IV. Skiagram of Ollier's disease. Cartilaginous deposits are confined mainly within the limits of the ends of the shafts of the bones.

In children there are certain conditions in which tumour-like masses of cells occur in the bones, very closely resembling neoplasms; I refer to certain affections of the reticulo-endothelial system, in which hyperplasia of the reticulo-endothelial cells of the bone marrow occurs, and in some of these conditions there are tumour-like masses of cells, loaded with lipoid, either in the flat bones or in the ends of the diaphyses of the long bones. In Schüller-Christian's disease, the main symptoms, exophthalmos and *diabetes insipidus*, are due to pressure on the orbit and pituitary gland. Until quite recently, most authorities actually called this condition osteogenic sarcoma. (See Figures I, II and III.)

Then, again, certain of the osteodystrophies are characterized by the presence of tumour-like masses of cartilage in the ends of the diaphyses. In Ollier's disease these cartilaginous masses are confined within the limits of the bone; and in hereditary deforming dyschondro-

plasia there are cartilaginous outgrowths as well. (See Figures IV and V.)

Hence, at the outset, one can see that the subject of bone tumours is a difficult one, as it is impossible at present to determine with certainty the constituents of bone from which some of these tumours arise, and also because there are many border-line conditions, particularly in children, forming connecting links between bone tumours and diseases of the blood-forming organs.

FIGURE V. Skiagram of a case of hereditary deforming dyschondroplasia. Cartilaginous deposits are seen in the ends of the shafts of the long bones, together with exostoses.

In studying bone tumours there are certain differences between the structure of the bones of children and adults which we must bear in mind. The presence of an epiphyseal cartilage in the growing bones of children tends for a time to limit the spread of a tumour, just as it limits the spread of osteomyelitis. In children under the age of seven years all bone marrow is red marrow. In adults the red marrow is confined to the vertebræ and flat bones. The change from red to yellow marrow in the long bones occurs at the age of puberty, although the upper ends of the femur and humerus retain their red marrow longer than the distal extremities of these bones, and longer than both ends of the more distal bones, so that the upper ends of femur and humerus are not uncommon sites of metastases from visceral growths.

DIAGNOSIS OF BONE TUMOURS.

It is sometimes exceedingly difficult to diagnose certain malignant tumours of bone in the early stages. As in the diagnosis of many other conditions, the radiographer can give us great assistance, and the pathologist's help must be secured to determine the nature of these tumours; but, unfortunately, a biopsy is not free from risk of spreading the disease and of hastening metastases, and so a biopsy is seldom

advisable. Hence we must frequently rely for the diagnosis on clinical evidence and X ray examination; and in many cases I think that a good detailed clinical history is of even greater importance than the report of either the radiographer or the pathologist.

There are certain bone conditions which are sometimes followed by malignant disease. Both bone cyst and Paget's disease are occasionally followed by sarcoma. Two such cases have occurred in my own experience within the last few years.

A female patient, aged fifty years, sustained a pathological fracture of the femur, and the radiographer reported a fracture through a bone cyst. Several months later an osteogenic sarcoma developed in the site of what was originally reported as being a bone cyst.

Possibly, in this particular case, the radiographer erred in the diagnosis of bone cyst.

Another patient, a male, aged forty-five years, was said to have Paget's disease of the upper end of the tibia, and three years later an osteogenic sarcoma developed in the site of the area, diagnosed after X ray examination as Paget's disease. The leg was amputated, and pathological examination confirmed the diagnosis of sarcoma.

It is sometimes very difficult to distinguish between chronic osteomyelitis and neoplasm by radiological appearance; and the records are full of cases in which osteomyelitis has been diagnosed on radiological examination, and in which the condition has proved later on to be either an osteogenic sarcoma or a Ewing's tumour. Occasionally the reverse has occurred, and limbs have been unnecessarily sacrificed for osteomyelitis. I do not wish to belittle the indispensable help that the radiographer can give us in these cases, but merely to emphasize that we must weigh all the evidence obtainable, and must not let the evidence obtained by X ray examination outweigh that derived from clinical examination. The pathologist may also have great difficulty in finding tissue upon which he can give a satisfactory report, when only small sections of the suspected tumour or fragments which have been curetted away are submitted to him for examination.

The following is the history of a case which caused me considerable difficulty in diagnosis.

A boy, aged nine years, suffered from pain in the thigh for several months. (See Figure VI.) There was an indefinite history of injury, but no history of an acute onset. He was a healthy-looking lad with normal temperature and pulse. A thickening of the femur could be felt, extending from just above the knee to the junction of the middle and upper thirds of the thigh, and there was tenderness over the same region. The X ray appearance suggested osteomyelitis; indeed, the three radiographers who saw the skiagrams were of the opinion that osteomyelitis was present. There seemed to be a definite involucrum, and some necrosis of the shaft. In one region definite layering of the newly formed bone could be seen, similar to that which is sometimes observed in Ewing's sarcoma; but such an appearance is not very uncommon in chronic osteomyelitis.

I advised a course of deep X ray therapy, and there was a definite lessening of the pain after this treatment, although during the subsequent week the patient's temperature became slightly elevated. The radiographer, however, reported that the response to radiation was not what would be expected in the case of a Ewing's tumour, and so he was definitely of the opinion that the condition was a chronic osteomyelitis. During the course of the deep X ray

treatment, a pathological fracture occurred, and several inches of the shaft of the femur seemed to be separating as a sequestrum. As there was still some doubt in my mind about the diagnosis, I decided to make an incision down to the bone and to secure some tissue for pathological examination, although I realized that if the condition proved to be sarcoma such a procedure might do harm. The bone was exposed and a small sequestrum was removed. Some soft greyish material occupied the medullary cavity, but there was no sign of any pus. A small section of greyish material was removed from the periosteal region, and a culture was made from the medulla. The culture grew *Staphylococcus aureus*, and no trace of anything like tumour cells could be seen in the section of the tissue removed. An enlarged inguinal gland had previously been removed, and sections of this did not reveal any evidence of neoplasm.

Hence up to this stage the clinical evidence of this case was in favour of neoplasm, and the radiological and pathological evidence was in favour of chronic osteomyelitis. The temperature became elevated for a week after the operation,

FIGURE VI. Skiagram of the femur of a boy, aged nine years. Layering of periosteum can be seen, also pathological fracture. No evidence of pus was found by exploratory operation at the end of three months. Radiographers consider the condition to be one of chronic osteomyelitis. A pure culture of *Staphylococcus aureus* was obtained at operation.

and the wound broke down and discharged a little pus, which ceased after one week. Moreover, the swelling diminished slightly in size, so that, although the evidence so far favoured osteomyelitis, it is impossible to be quite sure of this diagnosis, especially as Kolodny has reported a case in which a child was treated for two and a half years as suffering from osteomyelitis, as all the evidence obtained after several operations and the results of X ray examinations were in favour of such a diagnosis. But the child died two years and ten months after the first symptom, and the final diagnosis was sarcoma.

The history of another case which has caused me considerable difficulty and in which the diagnosis is still doubtful, is as follows:

M.O., aged six years, a female, suffered for five weeks from intermittent attacks of pain in the region of the hip joint. X ray examination revealed an abnormal appearance in the region of the great trochanter, which the radiographer reported as being either due to chronic osteomyelitis or tumour. The absence of any inflammatory reaction about the area was in favour of tumour. The child had no fever, but tenderness was present over the upper portion of the thigh. A von Pirquet test gave a positive reaction. A course of deep X ray treatment has brought about so much improvement that there is very little evidence of any trouble at the present time. Soon after X ray treatment was commenced a metastasis appeared in the neck of the scapula, and this cleared up completely after X ray treatment. The child now appears quite well, having increased

greatly in weight, and the question is whether any recurrence of the trouble is to be expected.

Again, we sometimes see cases in which there is an obvious tumour of a particular bone, and after taking into consideration the clinical history, the X ray report and the pathological evidence, we make a diagnosis, only to find later on, perhaps after a *post mortem* examination, that the tumour we have been investigating is a metastasis, the primary growth being in adrenal, kidney or other organ.

Sometimes, even after a *post mortem* examination, it is difficult or impossible to say whether the growth we have been observing is primary or secondary. The history of such a case, which was under the care of Dr. Wilfred Vickers, is as follows:

M.P., a male, aged three and a half years, was admitted to hospital with a large tumour of the mandible, a section of which had been removed at another hospital and diagnosed as a round cell sarcoma. X ray examination suggested that it was an epithelial adamantinoma of the mandible. Another biopsy was done; this showed an irregular growth of cells of epithelial type in alveolated stroma, and was not considered typical of an adamantinoma. The tumour grew rapidly, and deep X ray therapy was instituted; slight reduction in size followed this treatment. A few weeks later the child complained of abdominal pain, and laparotomy revealed nodules in the mesentery and bowel wall.

Post mortem small nodules of growth were found in heart and pericardium and also in the bowel in the ileo-caecal region. The mesenteric glands were infiltrated with growth. The whole of the retroperitoneal tissues was filled by a nodular mass which involved the kidneys and suprarenal glands. Several subperiosteal growths were found in the skull. Microscopically the growth was seen to be composed of small round cells with clear nuclei and little cytoplasm, and here and there a macrophage.

Dr. Tidswell considered that the growth was a sarcoma. He was of the opinion that the presumptive primary growth in the jaw might have given rise directly to the growth in the ileo-caecal region, but thought that the other lesions in the heart, kidneys and calvarium indicated blood dispersal.

CLASSIFICATION OF BONE TUMOURS.

Tumours of bone may be classified into innocent and malignant, and the latter into primary and secondary, as follows:

A. *Innocent*: Osteoma, fibroma, chondroma, giant-cell tumour.
B. *Malignant*:
   1. *Primary*: Osteogenic sarcoma, Ewing's tumour, multiple myeloma, periosteal fibrosarcoma.
   2. *Secondary*: Metastases from tumours of genito-urinary system, thyreoid and breast.

Innocent Tumours.

Osteoma (see Figure VII).—A true osteoma is a rare tumour, and may be cancellous or compact. The cancellous osteoma occurs in young children at the end of the diaphysis close to the epiphyseal line, and, as the bone grows, the distance from the epiphyseal line becomes increased. It is usually capped by cartilage, and is sometimes called an exostosis. The subungual exostosis is a variety of cancellous osteoma which grows from the dorsum of the distal phalanx of the big toe. The compact

osteoma is an extremely hard sessile tumour, usually found growing from the vault of the skull.

Chondroma.—Chondromata are found chiefly in the ends of the diaphyses of the long bones in young people, also in the metacarpals and phalanges and in the pelvic bones.

FIGURE VII. Showing an osteoma of the lower end of the radius.

Fibroma.—Fibromata of bone are rare. A fibrous polypus may grow from the nasopharynx, and a fibrous epulis is occasionally seen.

Giant-Cell Tumour (Myeloid sarcoma). — Giant-cell tumours (myeloid sarcoma) (see Figure VIII) develop in children and young adults, and may be found in any bone which develops in cartilage. The more common sites are the lower end of the femur and the upper end of the tibia. They may also be found in that portion of the mandible which develops in cartilage, namely, between the mental foramen and the symphysis. The centre of the bone becomes expanded, and the cortex is reduced to a thin shell. The X ray appearance is characteristic—the so-called soap bubble appearance. Although spontaneous fractures are common, giant-cell tumours are only locally destructive, and are not malignant. Most of them respond to X ray treatment, some few do not. Some authorities consider that they are inflammatory rather than neoplastic in nature.

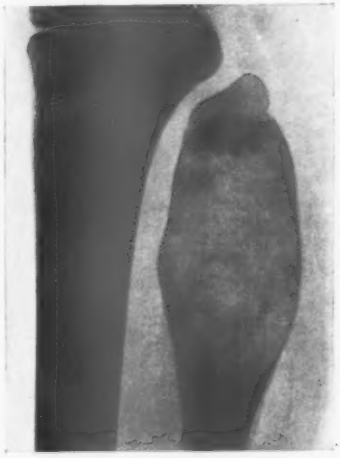

FIGURE VIII. Skiagram of a giant-cell tumour of the upper end of the fibula.

Epulis.—The common epulis is really a giant-cell tumour. Epulis arises from the alveolar dental periosteum of the deciduous teeth, generally the incisors, canines or bicuspids, and it hardly ever occurs at the site of the molars which make only one appearance. Epulis does not invade the bone, and, like other giant-cell tumours, is only locally malignant. Three of these cases occurred in the Children's Hospital in

the last couple of years. All occurred in the upper jaw, one in connexion with the incisors and two with the bicuspids.

Malignant Tumours of Bone.

Osteogenic Sarcoma.—The osteogenic sarcomata constitute the main group of primary malignant tumours of bone. They usually occur about the second decade, and are more common about the age of fifteen years. Their most characteristic site is the metaphysis of the long bones, and the rapidity of their spread along the shaft depends on the site of origin: the nearer the origin to the medulla, the more rapid will be the spread along the shaft. The epiphyseal cartilage tends to limit their spread towards the joint. The tumour gradually breaks its way through the bone cortex, at first pushing the periosteum before it, and then gradually penetrating it to reach the subcutaneous tissues. Usually the old cortex can be seen passing through the tumour mass, and this fact is of diagnostic importance. Spontaneous fractures are common, and metastases occur in the lungs. The consistency of the tumour varies, in some being very soft, in others bony. These tumours arise from the ancestors of cells which under normal conditions eventually become osteoblasts, so that an osteogenic sarcoma is really an osteoblastoma. In these tumours we find cells at different stages of development; some are simple spindle cells, others have developed a little farther to the mucoid stage, others may be cartilage, and some have developed to the true bone cell stage; hence the varying consistency of the tumour, according to the preponderance of the different types of cell.

Pain in osteogenic sarcoma may precede the swelling by weeks or months, and when the periosteum is involved it may be agonizing. The symptoms are not unlike those of a subacute or chronic osteomyelitis, and the presence of a moderate or even a high temperature is not uncommon. The X ray appearance is usually characteristic, as the gross structure of the tumour mass consists of stalactite-like rods of osseous tissue, arranged like the spokes of a wheel perpendicular to the long axis of the bone, giving the so-called sun ray appearance; or like fluffy trabeculæ spread beneath the periosteum on top of the cortex of the involved portion of the bone. These tumours are not radio-sensitive, so that the application of deep X ray does not reduce their size.

In the hospital records there are three examples of this kind of tumour.

M.H., aged twelve years (see Figure IX) had complained of pain in the left knee for about one month. A swelling was then noticed in the lower end of the femur just above the knee joint; this increased rapidly in size. The skiagram showed the typical appearances of an osteogenic sarcoma, and amputation was performed through the upper third of the thigh. No secondary deposits could be seen in the lungs. Dissection of the tumour showed that it was a fusiform growth, involving the lower third of the femur, and the bone marrow showed massive invasion. Microscopical sections showed the histological structure of a spindle-cell sarcoma.

F.B., aged twelve years, had an osteogenic sarcoma in the lower end of the tibia. In this case there were secondary deposits in the lungs and at the lower end of the femur.

C.H., aged nine and a half years, a patient under the care of Dr. Wilfred Vickers, complained of limping for three months. He had night starts and severe pain in the left thigh. There was a vague history of an injury to the leg. The patient's temperature was normal. The von Pirquet test gave no reaction. X ray examination revealed marked osteosclerosis, with great irregularity and some absorption of the neck of the femur, suggesting osteogenic sarcoma. The child developed scarlet fever, and was transferred to the Coast Hospital. Three months later the growth had extended into the joint and had involved the acetabulum. Metastases were seen in the lungs and in the third rib (see Figure X).

FIGURE IX. Skiagram from M.H., aged twelve years, of an osteogenic sarcoma of the lower end of the femur. Fluffy trabeculæ are seen, and the shaft of the bone passes through the mass of newgrowth.

Ewing's Tumour. — The variety of sarcoma known as Ewing's tumour is said to form about 10% of all bone tumours. It affects mainly children and young adults; 80% of the cases occur before thirty years, and most of these occur between the ages of five and fifteen. It generally grows from the middle of the shaft, thus differing in this respect from osteogenic sarcoma. Trauma is said to be a factor in its causation. The bones most frequently involved are tibia, humerus, femur, fibula, and clavicle, in that order. Of the small bones, the *os calcis* is most frequently involved, and occasionally the ribs, vertebræ, mandible and skull. It is three times more common in males than in females.

The origin of these tumours is believed by Ewing to be the perivascular lymphatic endothelium. Owing to the location of the tumour towards the middle of the shaft, joint involvement practically never occurs. When an apparently primary growth is under observation, X ray examination may reveal similar growths in other bones; the tumours seem to begin simultaneously in numerous areas, and there seems to be some doubt whether true multiple primary involvement is present, or whether these other growths are metastases. The fact that one may get extensive skeletal involvement with relative integrity of the viscera rather favours the theory of multiple origin and not skeletal metastases.

Pain in Ewing's tumour is generally intermittent, thus differing from the pain of osteogenic sarcoma. The recurrent attacks of pain may be associated with quite high fever and a moderate degree of leucocytosis. These attacks of pain and fever may be present for many months before

a swelling is noticed, and so an erroneous diagnosis of osteomyelitis is commonly made.

Months or even a year may elapse before pulmonary metastases reveal themselves and secondary tumours in other bones appear, the patient meanwhile becoming greatly emaciated and anæmic. Metastases in the regional lymph glands are not uncommon, although they do not occur so frequently as metastases in the skull and other bones.

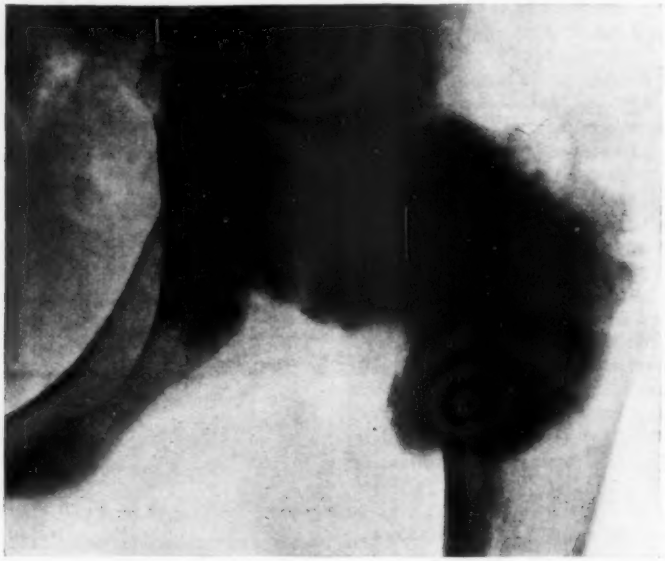

FIGURE X. Skiagram from C.H., aged nine and a half years, of an osteogenic sarcoma of the upper end of the femur. In this case the hip joint became invaded and the innominate bone was later involved. Metastases occurred in the lungs.

The X ray appearances are usually characteristic. Unlike osteogenic sarcoma, in which X ray examination usually shows the shaft of the bone passing through the tumour, in Ewing's sarcoma the bone is slowly displaced by tumour tissue, its absorption being accompanied by a widening of the shaft. The new bone is laid down parallel to the shaft, giving the so-called onion layer appearance in the radiograph. A diagnostic feature of great importance is that these tumours are radio-sensitive and so diminish in size after the application of deep X ray therapy; this is of more importance than a biopsy and pathological examination, particularly as an incision may be followed by fungation of the tumour. Spontaneous fracture is not common in Ewing's tumour; at any rate, not until the late stages.

There have during the last few years been several examples of this tumour in the Royal Alexandra Hospital for Children.

B.G., a female, aged thirteen years, fell and injured her knee (see Figure XI). Pain persisted for seven weeks; then a swelling was noticed over the lower end of the left femur. After X ray examination a diagnosis of Ewing's tumour was made, but as the blood gave a positive reaction to Kahn's test, the child was given antisyphilitic treatment as well as deep X ray therapy. After this treatment the tumour became smaller and the pain disappeared. Later amputation was performed.

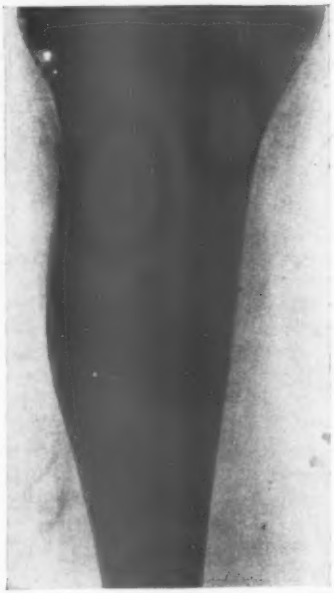

FIGURE XI. Skiagram from B.G., aged thirteen years, showing a Ewing's tumour. The shaft of the bone was displaced by the growth. Layering of the periosteum can be seen.

Multiple Myelomata.—I can find no instance of this disease amongst the records of the Royal Alexandra Hospital for Children, although it is said to occur occasionally in children. In cases which I have observed in adults, early anæmia and tenderness of the bones have been present, and spontaneous fracture has occasionally been the first evidence of the disease. It affects the red marrow bones in adults, and almost invariably the patients are over the age of forty years.

Chloroma.—Chloroma is a very rare tumour, and again I can find no record of a case having occurred in the Royal Alexandra Hospital for Children during the last ten years. This condition forms a connecting link between leuchæmia and tumour growth, being a malignant tumour with a blood picture resembling that of acute leuchæmia. It is commonest in children and in males below twenty years of age. The primary tumour is situated in the marrow of the long bones, and secondary growths occur in the bones of the face and cranium; exophthalmos occurs, being due to a periosteal growth in the orbit. Green tumours are also found in the sternum, ribs, spine and pelvis. The spleen and lymph glands are enlarged throughout the body.

Periosteal Fibrosarcoma.—Occasionally fibrosarcomata are met with growing from the fibrous or outer layer of the periosteum and not from the bone-producing cambial layer. These tumours are of the spindle-celled variety, the cells resembling fusiform fibroblasts. While these tumours do not invade the bone, they may cause its absorption or erosion as a result of pressure, and, unless such erosion has occurred, it may be impossible by radiographic examination to demonstrate any connexion with the bone; even at operation they may be difficult to distinguish

from a fascial sarcoma. Very few cases have been reported in the literature, and I cannot find any record of a case having occurred in this hospital. With regard to the age incidence of this variety of sarcoma, Kolodny[1] states that there does not seem to be any definite relationship between the incidence of the tumour and age. He states that he has observed one case of extraperiosteal sarcoma of the tibia in a new-born child, and adds that the child was alive and well ten years after amputation of the thigh.

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CERTAIN ASPECTS OF THE BASIC PATHOLOGY OF GOITRE: IODINE DEFECTS, COMPENSATORY ADJUSTMENTS, "MASS EFFECTS".

By A. M. DRENNAN,
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THE pathological changes found in the thyreoid in most, if not all, forms of goitre arise ultimately from a deprivation of iodine. An exception is sometimes made in the case of primary Graves's disease, but whether this condition may not also come under the general heading of iodine deficiency can be decided only by further study. It is true that thyreoid changes, approximating to the goitrous changes in the human, have been produced in animals by various experimental means, such as injection of anterior pituitary extract, feeding with certain of the Brassicas, and giving diets deficient in vitamin, especially if badly balanced as regards protein, fat and carbohydrate. In these experimental forms of goitre iodine will prevent that due to the action of the Brassica, though not, apparently, those forms due to pituitary extract or bad or deficient diet.

In the human subject the only clear relation of cause and effect is seen in the endemic area where iodine intake is deficient and goitre results. In the so-called sporadic cases it is usually said that iodine intake is adequate, but it is unsafe to accept this general statement for an individual case, as even scurvy may arise in an individual living amidst a sufficiency of vitamin *C*, but not partaking of it. So, with the sporadic case, the area and the community may have adequate iodine, but the individual, from personal idiosyncrasy or taste, may not include the necessary iodine-containing substances in his or her diet. The other factors seen to be effective in animals, namely, pituitary stimulation or Brassica activity, may play a part in these individual cases, but, as yet, they have not been studied with sufficient thoroughness to clear up these points.

If we accept as a working hypothesis that iodine deficiency, however produced, is the chief factor in the production of goitre, it will help in some measure to explain the pathological reactions in the thyreoid which constitute what we call goitre.

In an attempt to compensate for the inadequate iodine supply the thyreoid increases its surface area and storage capacity. It becomes a diffuse "goitre" of "parenchymatous" or of "colloid" type. Incidentally it seems unfortunate that these terms are used to denote different phases, as both are, strictly speaking, parenchymatous, the so-called "paren-

chymatous" representing the active phase, the "colloid" the passive or storage phase of the parenchyma.

Such enlargements are compensatory and serve to supply the body with its necessary hormone. In the active ("parenchymatous") phase there is a very low iodine content in the thyreoid as little or no storage is present, the hormone being produced and at once passed into circulation; in the passive ("colloid") phase the iodine content is higher, but still lower than the average normal. Giving iodine increases such content in the "colloid" and induces colloid storage in the "parenchymatous". These enlargements may never produce clinical disturbance, and may be discovered only by accident, or they may be large enough to be unsightly or even to cause pressure symptoms. The enlargement is more likely to persist and to increase in the female owing to recurrent stresses, such as those of menstruation and pregnancy, in both of which iodine is lost—in the former in the menses, in the latter as extra hormone required for the fœtus.

It is such goitre that ought to be prevented by a diet containing adequate iodine. Or the goitre having begun—being incipient—it may be arrested by iodine addition to make good the deficiency. In the latter circumstances a danger may arise: the gland has increased to balance a low intake of iodine, and if such a gland has already enlarged considerably and is suddenly given an excess of iodine, it becomes overcharged at a high iodine concentration and may cause "toxic" effects, as its size—as regards iodine content and hormone production—is now out of proportion to the size of the individual possessing it. It is this "mass effect" which is apt to be overlooked, which is seen in all forms of goitre and which is a large factor in the development of "toxic" goitre from a previously "simple" goitre. It is the mass effect which the surgeon corrects when he pares down the toxic goitre gland to correspond to the size of its owner.

An enlarged gland such as that described is prone to secondary changes. Parts become overdistended, hæmorrhages are frequent, with resulting disorganization and fibrosis. Exhaustion of parts with atrophy is common, often accompanied by lymphoid collections giving the so-called "lymphadenoid" goitre. Fibrous replacement of the atrophied parts occurs, and hypertrophy of less stressed parts, giving various forms of "nodular" goitre. The nodules may be of the "active" (hyperplastic: parenchymatous) or of the "passive" (colloid: storage) type. Only histological examination can identify these properly. All combinations of the active and passive types may be present. It is this variation that helps to explain the different responses to treatment in different glands. The colloid nodules, when exposed to iodine, store colloid richer in iodine; the hyperplastic nodules at first seem to continue in the active state, but in time may involute to the colloid form. This is probably the reason why some "toxic adenomatous" goitres respond to iodine favourably and some unfavourably.

The nodular goitre may be "simple" or "toxic", or may change from simple to toxic. The differences depend on the mass effect, which

again depends partly on iodine intake, partly on metabolic demand, and partly on amount of secondary changes. A large goitre may be merely a mechanical nuisance, a relatively small goitre an over-functioning menace.

The term toxic adenoma is commonly given to those nodular goitres with toxic symptoms. It is not possible either by macroscopic or microscopic examination to say what is a toxic and what a non-toxic goitre. It is necessary to know the total amount of the goitre, its iodine content in its several parts, as well as its histological structure, before an opinion can be given with reasonable accuracy as to its toxicity or otherwise. A true adenoma can occur in the thyreoid, as in any other gland, and it may function and thus produce hyperthyreoidism or toxicity from the increase of acting gland tissue. But such a true adenoma is usually single, and is well defined and usually associated with an otherwise normal gland. The usual toxic adenoma is a compensatory nodule, hyperplastic or colloid, or mixed, and is usually multiple. Such nodules represent the continued attempt of the thyreoid to utilize deficient raw material—iodine—for the hormone required for ordinary needs. More expenditure of energy demands in an already changed gland more hormone, more thyreoid activity, more goitrous change. Lessened energy expenditure still leaves a larger gland or more nodular gland for the unaltered size of the individual.

Spontaneous cure is possible from degenerative changes occurring in the excess thyreoid tissue; indeed, hypothyreoidism may so result. The more usual event is, however, that the gland, having originally enlarged and having become either a diffuse or, more commonly in older cases, a nodular goitre, is later associated with toxic symptoms. The later conditions—menopause, lessened activity, infection *et cetera*—upset the previous balance and the gland, being now too big for the individual, produces a hyperthyreoid state. The only adequate treatment for this phase is to trim the gland to fit the patient—to reduce its mass effect, as already indicated. It is to be remembered that in operations on the enlarged pathological gland some glandular tissue is left, and that the remnant is frequently of similar type to that which has been removed. Also, as seen in laboratory animals, removal of a large part of the thyreoid encourages the remainder to proliferate. To keep this proliferation within bounds and to encourage the "storage" type of gland, iodine in sufficient concentration is required. Omission of this may result in hyperplasia and overgrowth of the thyreoid, even approximating to its former size. Thyreoid extract in proper amount might act as an arrester of hyperplasia also by removing the need for activity of the gland tissue.

In the so-called primary Graves's disease the symptoms often are abrupt, with no previous warning of thyreoid disorder. Pathologically we find a diffuse hyperplastic gland, often with no old-standing change. With exhibition of iodine, such a gland usually involutes to the colloid, storage, stage. Unfortunately, we do not know in such cases what the

gland was like before the appearance of symptoms, and we are usually quite unaware of the actual iodine intake or requirement of the individual. Although the addition of iodine induces colloid involution and reduces the pouring out of hormone, it results in a relatively large gland now filled out with colloid of relatively high iodine content. The basal metabolic rate of the individual is reduced, but does not come down to normal level, and, unless the excess of thyreoid tissue is reduced surgically, the individual is in much the same state as is a person with a simple diffuse colloid goitre to whom iodine has been given. It is now a large colloid goitre with a high iodine content, and thus capable of issuing to the circulation more hormone than the size of the individual requires. In minor grades of primary Graves's disease it is true that, owing to the small increase of thyreoid tissue, rest and iodine may bring the gland to a reasonable balance for the size of the individual, but such a patient is ever prone to suffer from hyperthyreoidism if the gland is called upon unduly.

The pathological picture is, of course, complicated by all the secondary effects of increased metabolic rate—cardiac damage, stimulation of other endocrines *et cetera*, the intricacies of which are out of place in this note, which deals primarily with the goitrous thyreoid change. For the same reason malignant disease is omitted.

THE PROBLEM OF THYREOTOXICOSIS.

By HUGH R. G. POATE,
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THYREOTOXICOSIS has lately received a good deal of attention. Many articles dealing with it have appeared in the journals during the last two or three years, and it was discussed at the largest combined meeting at the annual meeting of the British Medical Association in Melbourne in September, 1935. In this discussion the clinical features and treatment of the common types of the disease were emphasized, but many difficulties and problems were left untouched, for the main object of the discussion was to interest the general practitioner and not the specialist. In this paper I intend to consider the condition from the point of view of those with a good deal more than an elementary knowledge of it.

TERMINOLOGY.

Lord Horder has expressed the view that the term thyreotoxicosis is not altogether satisfactory, as it postulates a basic fact which has never been established. We have learned that the condition embraces many more types than those listed under the classical description of Parry, Graves and Basedow, so that their names as applied to the disease are now of historical interest only.

The term "hyperthyreoidism" presupposes a condition due to an excessive amount of normal secretion, of which thyroxin is the most important constituent. In my opinion, it is a condition commonly seen in many young or youngish women prior to menstruation, and, with other physiological reactions of the gland, in males as well as females.

One of the most pithy descriptions of the thyreoid gland that I have heard is that it is "the gland of fight and fright". Hypersecretion increases the speed of living; all our tissues are keyed up to give of their best, so that we are filled with a sense of well-being and wakefulness, the flexibility and elasticity of our mental and physical processes are stimulated, and we feel, think and act clearly and quickly. Fortunately for us all the demand for thyreoid hypersecretion is made only occasionally. Apparently the adrenals supply the preliminary energizing factor for any sudden critical call on the individual, until the more slowly-acting but much longer-lasting thyreoid secretion can come into play. But constant and excessive demands on the thyreoid gland, as on all other tissues, may lead to degenerative changes in its secreting cells and to microscopic changes in the gland, so that dysthyreoidism or thyreotoxicosis may result. But we must not lose sight of the fact

that during these periods of over-stimulation all bodily tissues share in the flogging, and breakdown may occur in regions other than the thyreoid gland, as is instanced clinically by auricular fibrillation, glycosuria, alimentary crises, acute mania *et cetera*. One patient with hyperthyreoidism remarked recently that at the start of a wave it was a grand feeling, but the worst part came when you felt you could not stop!

It is interesting to note that one milligramme of thyroxin produces an increase in the basal metabolic rate equivalent to 2·8% of its normal value, and that this increase is maintained for several days. In the normal individual the daily consumption of thyroxin is probably less than 0·4 milligramme, and the total amount functioning in the body is only 8 to 15 milligrammes.

These cases of hyperthyreoidism are frequently associated with sympathetic imbalance, are eminently curable by suitable medical treatment, and should not be classed among the thyreotoxic states.

The titles "toxic goitre" and "exophthalmic goitre" are limited in their application, as in many cases there is no goitre, the gland may even be smaller than normal, and in most of the cases now seen there is very little, if any, real exophthalmos.

We thus come back to the term "thyreotoxicosis" as being for the present the most suitable title for the symptom complex under discussion. That there is a real toxic secretion seems definite on clinical grounds, especially when one considers those peculiar cases in which positive signs and symptoms of myxœdema are present along with equally positive signs and symptoms of thyreotoxicosis.

ÆTIOLOGY.

As with many other ailments there does not seem to be any one or even a series of definite causative factors. Dunhill divides the factors into three sections: (i) the stimuli, (ii) the thyreoid gland, and (iii) other body tissues. He points out that each of these is capable of many variations, and that different reactions occur in separate individuals according to the variations and predominance of the several factors; thus he accounts for the multiplicity of clinical manifestations in what he believes is basically a single pathological entity.

The youngest patient I have seen with thyreotoxicosis was a child of six years, who presented the classical picture of acute Graves's disease, and I have seen several of from twelve to sixteen years of age. What was the stimulus in these cases?

It is said that the sex epochs play an important part. The only sex epoch that I am sure of as a factor is the menopause, and I believe that a close analysis of the symptoms usually complained of as accompanying the "change of life" will show many of them to be due to thyreoid imbalance. If sex epochs are a big factor, why do we not see many more cases at the age of puberty? Or is it possible that the continued hypersecretion associated with menstruation may lead to dysfunction of the thyreoid gland over a period of years? If so, what

is the sex epoch which influences the male, for the disease is much commoner in males than is generally supposed?

Some of the most acute thyreotoxic conditions that I have seen and most difficult to control have been induced by the taking of large doses of thyreoid substance for weight reducing, and followed on a definite period of true hyperthyreoidism.

The bogy of focal sepsis has been raised and given pride of place by some, but to those of wide experience it is found that if focal sepsis is a factor at all, it is a secondary and not a primary factor, for it acts rather by aggravating a preexisting condition than by initiating the disease. It is in the same category as any prolonged or exhausting illness which makes a long continued demand on thyreoid activity. If focal sepsis is present, most of us are agreed that it is in the best interest of the patient to deal with the thyreotoxic condition first, and then to clear any focal sepsis, not *vice versa* as is so often advised.

There is no doubt that psychic trauma, especially when associated with physical insult, is a stimulus to the onset of the disease, but chiefly when the thyreoid tissue is in a state of imbalance or exhaustion.

The other endocrine glands, especially the adrenals, the parathyreoids, the ovaries, the pituitary gland and the islets of Langerhans in the pancreas, play some part in the disease, and may modify it in many respects. I have seen a fulminating thyreotoxic condition associated with acromegaly clear up with no other treatment than deep X ray therapy to the pituitary region.

It seems to me that the chief ætiological factor is the condition of the gland itself, especially if it is the seat of multiple adenomata; and in many cases we can elicit a history of thyreoid enlargement in early childhood which apparently cleared before the age of twenty years.

Before leaving the question of ætiology I must quote Dunhill, who states:

> I believe that the stimuli which bring about the disease are situated without the gland, and that these stimuli affect the gland, causing such change in its structure and function that its secretion is altered and consequently other organs are intoxicated by its altered secretion.

DIAGNOSIS.

Diagnosis is easy in the greater proportion of established cases, but we are learning to recognize the earlier manifestations of the thyreotoxic state. Among early signs and symptoms are those of over-stimulation of the sympathetic nervous system; and I may mention in particular a state of excitability and nervous instability evidenced by irritability and outbursts of temper, flushings, rapid breathing, fluctuations in weight, attacks of palpitation, periods of physical exhaustion, sweatings *et cetera*. How often does one hear that the patient has always been "highly strung" and even classed as being neurasthenic? For some reason or other I have found the earlier stages of the condition more often overlooked in males than in females. If the condition is suspected, it is now easy to obtain confirmation by an estimation of the basal

metabolic rate; but a patient should not be sent for a test during a period of remission or after a course of sedatives and iodine, or the surgeon may be misled by a reading that is within normal limits. There is no doubt that during the last few years there has been a great increase in the number of cases. This is not merely a matter of improved diagnosis, but seems to be an aftermath of the hectic years of plenty, followed so quickly by the black depths of the years of depression—in fact, a striking example of psychic stimuli as a factor in ætiology.

PROPHYLAXIS.

Believing, as I do, that the main factor in ætiology is the state of the thyreoid gland, I would urge the importance of early removal of all adenomata or cysto-adenomata found, irrespective of the age period. I know of no other type of so-called simple growth in which the patient is so often told by the family practitioner, or even by the specialist physician, to leave it alone as it is doing no harm; yet with no other simple growth is the potentiality for harm so great as with adenoma of the thyreoid gland. Quite apart from the question of the onset of hyperthyreoidism or thyreotoxicosis, I may mention that in nearly every case of carcinoma of the thyreoid that I have seen there has been a history of a lump in the gland for years.

All thyreoids that are the site of multiple adenomata should be subjected to subtotal excision if they are extensively involved, or in minor cases to excision of the obviously affected areas. If symptoms of hypothyreoidism do supervene, they are easily controlled by a daily dose of thyreoid substance, which, after all, is a small price to pay for good health.

To all those patients showing signs of hyperthyreoidism as distinct from the toxic condition, the position must be carefully explained, and they must be content to live within the ambit of their own normal thyreoid output. In periods of excitement and exaltation, or prior to the menses, small doses of iodine given once or twice a day for one to three days generally work like a charm, but most patients require bromide or other sedatives as well. By a careful regulation of their lives it is possible to carry these patients through the sthenic years of life until they reach the years of discretion, which seem to develop coincidently with recession of thyreoid activity. Special care should be taken of these people during and just after the menopause, as they are particularly liable to develop a toxic state leading to some form of cardiac breakdown.

It has been suggested that in these cases the thyreoid gland has become adjusted over a number of years to a low iodine intake. When the increase of hormones usually occurring at the menstrual period is no longer required, the system generally becomes overcharged for the body needs and a toxic syndrome ensues.

TREATMENT.

Patients with true hyperthyreoidism respond well to rest, to sedatives and to freedom from excitement, worry and noxious stimuli.

Small doses of iodine may be given for periods of one to seven days. In the established thyreotoxic states when operation is indicated, iodine should not be given until the patient is in hospital and undergoing pre-operative treatment. In doubtful cases iodine therapy is admissible in minimal doses, and only for very limited periods, not exceeding three weeks. Experience has taught me that the smaller the dose that is required to bring the patient on to a stable level, the better the result for that individual. For the sake of obtaining a uniform standard, I employ only Lugol's solution, and even in the worst cases seldom find it necessary to give more than three to five drops three times a day.

I have seen one patient who had been given ten drops after each meal for nearly three years, but I am glad to say that most patients coming to me now bring a letter from their doctor, saying: "I have given no iodine as yet", or "I tried iodine for ten days, and the response was so favourable that I suspended it pending the institution of pre-operative treatment". The main points to be watched in giving iodine are: (i) the slowing of the pulse rate, (ii) the amelioration of symptoms, (iii) the hardening of the thyreoid. If these occur, it is certain that there will be a coincident drop in the basal metabolic rate and that early operation will be safe.

In the very early cases, especially of the frank Graves's disease type, medical treatment is the best procedure until a period of chronicity or of stability in symptoms has been reached. The best sedative is bromide, aided in some cases with small doses of a barbiturate, but in my experience these patients do not stand barbiturates well, and they should never be given in large doses, especially for operative premedication. I am also opposed to the use of atropine or belladonna because of their action on an already overtaxed sympathetic nervous system.

The worst risks are presented by those patients who are in an ascending wave of thyreotoxicosis, but once a stable plateau has been reached the surgeon should be called in.

Young people with uniform, large, soft glands are the ones who seem to respond best to X ray therapy.

The onset of auricular fibrillation is an urgent indication for surgical treatment, and occurs only in established thyreotoxicosis. The inference is that no medical man treating a case of thyreotoxicosis should allow fibrillation to develop.

Digitalis in the presence of a thyretoxic factor loses much of its potency in the control of auricular fibrillation, and chief reliance is to be placed on measures directed to control the toxic disturbance. Digoxin, however, may be very helpful.

In all these cases it is well to remember that an increased metabolic rate means a big drain on the biochemical processes of the body tissues, with a corresponding increase in waste products; as a result, the alkali reserve becomes depleted owing to the greater need for buffering the increased acid content of the blood stream, and thus we have a definite tendency to a true acidæmia.

If patients in this condition are subjected to general anæsthesia and operation there is a grave risk of a fatal crisis, and I am sure this was a big factor in those severe post-operative reactions that we used to see before the necessity for a careful pre-operative preparation was recognized. Accordingly, I always include in treatment a fairly liberal amount of alkali, and find that bicarbonate of soda is the best of all for general use. Also, I attempt to increase the glycogen storage by giving glucose.

Another important point about the operation is the time factor; unless there is a feeling of certainty that the operation can be completed within forty-five minutes, it is wiser to stop at a hemi-thyreoidectomy and to complete the operation on the other side ten to fourteen days later. I would urge surgeons beginning thyreoid surgery to bear this in mind, and to plan the operation in two stages until they achieve the manual dexterity and operative technique which will enable them to embark on the complete operation.

Some of the worst risks are presented by the large diffuse goitres of acute onset, and next to these I put the large flabby thyreoids, the site of multiple adenomata in the middle aged person.

The hyperiodized patient presents a problem; I think it best to rely on alkalis, sedatives, rest and diet for from four to six weeks, and then to resume minimal doses of iodine. If the patient does not respond, a course of deep X ray treatment may help to ameliorate the toxicity to a sufficient extent to justify at least ligation of the superior thyreoid vessels under a local anæsthetic. Later, further operation may be undertaken, preferably in two stages.

I have seen two patients undergoing deep X ray treatment develop acute mental disorder and die. They were "bad risks", and whether death would have occurred in any case one cannot say, but I rather feel that the X ray treatment influenced the fatal termination.

Now we come to the common question as to how much tissue should be removed. In order to get down to a definite basis, I have for many years removed the whole of the right lobe, the isthmus, the pyramidal lobe if present, and varying portions of the left lobe, depending on (*a*) the degree of toxicity, (*b*) the size and condition of the gland, (*c*) the age of the patient, and (*d*) the difficulty of access. As a rule, it is necessary to remove more gland in males than in females, in the very toxic cases, in the multiple adenomatous types, and in those with severe cardiac disturbances. In fact, in the last-mentioned cases I feel that a practical ablation of the gland is the best thing to do, and it is always better to take too much than too little, as any tendency to myxœdema can be controlled easily by a judicious daily dose of thyreoid substance, whereas any toxæmia persisting threatens the individual with ultimate breakdown, just as surely as does the original condition, although more slowly.

Post-operative reaction is negligible in cases wisely selected, properly prepared and skilfully operated upon. Occasionally a risk

must be taken, and then glucose, bicarbonate of soda and saline solution are given *per rectum*; frequent doses of morphine are given, and possibly intravenous injections of sodium iodide and, if necessary, intravenous injections of digoxin.

I cannot leave this question without again quoting Dunhill:

> No surgeon should operate on toxic goitres who is not prepared to remove enough thyreoid tissue to effect a cure, otherwise he will have sufficient bad results to render a serious dis-service to surgery.

At times, especially in young people, the gland tissue left at operation may hypertrophy and further operation becomes necessary, but in mild cases I think a few applications of X rays will achieve the desired result of a stable balance.

Sometimes I am asked what is the result in those who are either unable to afford the luxury of complete invalidism and medical treatment, or who do not respond to it and yet refuse the aid of surgery. My reply is that there are several possibilities: (i) They may become burnt out wrecks, with more or less permanent invalidism. (ii) Auricular fibrillation and congestive heart failure may close the picture. (iii) Acute mania may develop. (iv) Glycosuria may ensue. (v) Death may result from general exhaustion. (vi) A fortunate few may establish a state of equilibrium and tolerance which will permit them to lead a shortened life of restful ease.

HYPOPARATHYREOIDISM.

Before concluding, I must mention one difficulty occasionally met with after operation—the onset of hypoparathyreoidism. This is evidenced by a feeling of intense weariness and exhaustion, along with a peculiar firming and stiffness of the muscles, often with cramps, and in extreme cases with tetany, swelling and tenderness of the breasts and the development later of follicular cysts in the ovary, with menorrhagia and other menstrual upsets. As a rule the condition is transitory, and if it is recognized early and treated promptly, it will clear on the exhibition of large doses of vitamin *D* or the injection of parathormone and possibly the giving of parathyreoid extract, although I have very little faith in the last mentioned.

I have also seen this condition develop as a result of X ray treatment. One of the worst cases that I have met with was in a woman who had it as a result of X ray treatment prior to operation.

CONCLUSION.

As a final remark, I would say that I feel that the whole question of ætiology and treatment of thyreotoxicosis is still in a state of flux, but I am sure that in the light of our present knowledge the quickest and most certain method of restoring the patient as nearly as possible to normal is by operation carried out with adequate pre-operative precautions by a competent surgeon.

ILEO-CAECAL ADENITIS.

By Arthur E. Brown,
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All surgeons have had the experience of diagnosing appendicitis in children on fairly definite symptoms and signs, and have found at operation that the appendix itself looks disappointingly normal, and that there is a mass of enlarged glands in the ileo-caecal angle. However, that the series of circumstances leading up to this finding represents a clinical entity in itself, does not seem yet to be generally recognized. I have been interested in these cases, and propose to present my own experience with them, over a number of years, in the hope that discussion of them may lead to a more general recognition of ileo-caecal adenitis as an alternative diagnosis to appendicitis in children.

The condition may occur in either an acute or in a subacute or recurrent form, and may thus imitate either acute or "chronic" appendicitis. As with appendicitis, the two groups show somewhat different characteristics, though they naturally overlap to a considerable extent. I have had to deal with 23 cases of adenitis, of which 16 were of the subacute or recurrent type, and seven were acute. In the same period of time I have operated on 111 other children under eighteen years of age for appendicitis in all its forms, which makes the incidence of adenitis among children seeking surgical aid for symptoms suggestive of appendicitis 17·3%.[1] That percentage is certainly not negligible, and I have learned to pay careful attention to the history and clinical examination of such cases with the alternative diagnosis in view. Boys and girls seem to be about equally affected by adenitis, with some preponderance in favour of boys; and this is in striking contrast to the incidence of "chronic" appendicitis, for which girls are overwhelmingly more frequently operated on than boys.

Clinical Features.

The disease may be looked for in children between the ages of three and eighteen years. My youngest patient was four and a half. The oldest was just on nineteen when operated on, but had been watched through repeated attacks since the age of twelve. In the acute type, the child may be brought to operation during the first attack, as in these more virulent forms the inflammation persists and the condition remains menacing. I have seen one such attack last for eleven days before operation, with no sign of remission until operated on. In the

[1] Since preparing this paper I have operated upon 26 children under the age of nineteen years for symptoms of an appendiceal type, of whom seven (four boys and three girls) were suffering from adenitis.

milder form it is almost the invariable rule for a history of previous similar attacks of pain to be obtained, reaching back for months or years. The number, frequency and severity of the attacks vary in the descriptions given by the mothers. The duration varies from ten minutes to two days. In the attacks which I have personally seen, the rule seems to be for the pains and tenderness to persist for about forty-eight hours, with gradual subsidence. In view of the obvious supposition that bowel disorders may precede or be responsible for the adenitis, I have made careful inquiry into this in all cases, but have obtained no information to support such a theory, and in general it is not to be looked for.

Precedent general toxæmic symptoms, however, are of much greater diagnostic importance. In most of the seven acute cases they were well marked, being called by the parents "tonsillitis" in one case, "influenza" in another, "drowsy and feverish" in a third, and "feeling sick" in a fourth. This symptom was in all cases noted before the pains set in, in one case five days before. Among the 16 subacute cases, five patients gave a definite history of precedent malaise, and four showed evidence of decided toxicity during the attack, two having albuminuria, and one icterus. Two others were mildly toxic.

Vomiting is not a very marked feature of the attacks. It occurred only five times in these 23 cases, and in only one was it at all persistent.

The location of the pain varies, the most usual history being of generalized pains, which in about half the cases become localized later to the lower right quadrant of the abdomen. But this is not an invariable history, and the pains may be localized from the start, or may not be localized at all, without affecting the clinical diagnosis. The tenderness associated with it is much more important and definite. It is to be looked for close to the umbilicus, generally just to the right and below it. It is a definite diagnostic point that the spot of maximum tenderness is not the "McBurney's point" of appendicitis. Occasionally it may be both to right and left of the umbilicus, but I would regard the finding of maximum tenderness close to the umbilicus on the right side as very important for diagnosis. If along with the pains and this spot of tenderness there is absence of rigidity, I would regard adenitis as the most probable diagnosis. Absence of rigidity of the abdominal wall, in a case otherwise suggestive of acute appendicitis, should at once bring to one's mind the possibility that adenitis may be the cause.

It is not necessary for the establishment of the diagnosis that a mass should be felt. One was palpable in only three cases of this series, all three being of the acute type, in which the glands were conglomerate and intensely inflamed. The absence of a palpable mass in the less virulent cases is readily understandable when the enlarged glands are seen at the operation. It should none the less be looked for, and, if present, will probably be felt higher and more medial in position than the usual appendix-omentum mass of appendicitis.

The usual signs of inflammation—the state of the tongue, of the pulse and of the temperature, and the leucocyte count—are not of much

assistance in making a differential diagnosis. They are all, in general, on the mild side. I have seen in a subacute case a leucocyte count of 31,000 per cubic millimetre with 86% of polymorphonuclear cells, and one of the most acute cases, with palpable mass, with a maximum leucocytosis of 18,000 per cubic millimetre. It is most usual to find the leucocytosis to be under 20,000. The differential count also is generally that of a fairly mild inflammatory reaction on the whole, and is not of any great diagnostic value.

OPERATIVE FINDINGS.

The appendix, seen when a patient with adenitis is operated on, differs quite slightly, if it differs at all, from the normal. In half my cases it was recorded simply as "normal". In the others it was slightly more bulky than usual, with some evident congestion of the serous coat, giving it a pinkish tinge. The mucosa is normal to the naked eye, and the contents are generally semisolid fæces, or occasionally a stercolith. Examined microscopically, most of these appendices were reported on as normal or slightly congested, but with very little evidence of any active inflammation. In an endeavour to establish a supposition that the reddening sometimes seen represents a residual inflammation, and that the inflammation of the organ as seen at operation is in a subsiding condition, after having sustained an infection and infected the glands, I divided the cases into those operated on within three days of the onset of the symptoms, and those with a duration of longer than three days. I found no support for the theory. Cases of ten and eleven days' duration showed the slight reddening in just the same proportion as did the earlier cases; while an absolutely normal appearing appendix is as likely to be found on the first or second day of the attack as in the second week.

The last few inches of the ileum sometimes share with the appendix this slight congestive reddening. Nothing more definitely departing from the normal was seen; and in about half the cases there was not even this degree of change.

Only in two cases was any abnormality seen in the wall of the caecum or colon. There were in one family three children who showed identical acute symptoms within a few days of each other. In one of them the caecal wall and mesoappendix were densely œdematous; in another there was a large patch of œdema in the inner aspect of the hepatic flexure; while the third, in view of the operative diagnoses made in his brother and sister, was not operated on, and the condition settled down. These three cases had undoubtedly a common infective origin, and I regard them as being somewhat different from the other cases in the series.

Occasionally a small amount of free fluid was noticed in the peritoneal cavity. It was never great, and may on other occasions have escaped my notice. The peritoneum itself seemed to take very little part in the inflammatory process in the glands underlying it. Only in the two cases mentioned above with œdema in the large bowel was there

any œdema in the mesoappendix or of the peritoneum. This is in rather striking contrast with the œdema of the mesoappendix which is seen so frequently in cases of acute appendicitis. The peritoneum is usually stretched over the glands, and is not even conspicuously reddened. This fact has probably some bearing on the lack of rigidity of the abdominal wall which is a feature of the clinical picture.

The whole of the pathological picture disclosed seems to lie in the glands. The inflamed glands are those in the ileo-caecal angle, and along the inner border of the caecum, the chain spreading centrally along the ileo-colic artery. In six cases palpably enlarged glands were felt in the root of the mesentery, and in five definitely enlarged glands were visible in the mesentery of the lower part of the ileum. In one case I found an enlarged gland in the angle between the two common iliac arteries. In every case the gland chain was large enough to be immediately felt by the exploring finger. When discrete, the individual glands are about the size of French beans. In colour they are a reddish-pink. Except in those cases in which they had fused into a conglomerate mass, they were easily movable under their peritoneal covering, and, on this covering being incised, they readily extruded. On section of their capsule, the reddish gland substance bulged out. Microscopic section of a number of them revealed nothing more than a lymphoid concentration of round cells, and agar slope cultures of the scrapings from the cut surfaces of two of them, cut *in situ*, did not produce any bacterial growth.

CLINICAL COURSE OF THE DISEASE.

Since the positive diagnosis of adenitis is confirmed only by operation, all reported cases are shown as terminating by operation. It is likely that many patients suffering from infection of the glands have symptoms not severe enough to bring them to operation, and so never get into the records of adenitis cases. I have among my own records several histories of children in whom in the last few years I have made the diagnosis of adenitis, and deferred operation; but, the diagnosis not being proved, they cannot be discussed here. I have, however, been able to watch personally for years, both before and after operation, my patients submitted to operations. The course of events is apparently this: A child suffers infection of the glands, causing an attack of pain. Usually this is allowed to subside, which it does; but once the glands have become infected, the acute attacks of swelling have a definite tendency to recur, until finally the repeated attacks of pain lead the mother to seek medical aid. If, as often happens, the clinical picture is not quite that of appendicitis, if the tenderness has passed off or is placed round the umbilicus or even, as I have seen in one case, to the left of it, operation is deferred. The attacks are still repeated, and finally the child is operated upon and the appendix is removed. From that time onwards the attacks of pain cease, and the child remains in good health. I have watched the pre-operative course of one such child

for seven years, others for three or four years or less, and I am firmly of the opinion that until the operation of appendicectomy is performed, the attacks will inevitably recur. On the other hand, I have not seen any case in which attacks have recurred after the appendix has been removed. What would happen to the glands if the operation were not done it is difficult to say. The nature of the cases and the condition for confirming the diagnosis forbid discussion of that point. Whether the glands would become calcified, or whether they would gradually lose their susceptibility to the attacks of inflammation with succeeding years, is problematical. In the case in which operation was performed after a seven years' history, the glands were described as "some hard and calcified, some caseous, some acutely inflamed and œdematous". In other cases operation performed three or four years after the first attacks, revealed glands no different in appearance from those seen in early attacks.

DISCUSSION ON PATHOLOGY.

This brings one to the consideration of what are the pathological factors involved in this disease. I think one must rule out such static conditions as the "lymphoid hyperplasia" of some American writers,[1] and agree that the clinical picture is consistent only with an inflammatory process. And having said this, one immediately faces the question of the position which tuberculous infection occupies in the pathology. Ten or twenty years ago the tuberculous nature of the swelling would hardly have been questioned. Many would barely question it now. But microscopic and bacteriological examination of the glands by many skilled observers have repeatedly failed to reveal either tuberculous structure or tuberculous bacilli in them. Strombeck[2] regards as the most reliable guide in distinguishing tuberculous from non-tuberculous glands, the occurrence of calcification in them as shown in X ray films. He says that calcification can be observed in the films in from eighteen months to two years after the onset of attacks. He failed to prove any tuberculous origin in about 13% of some three hundred and odd cases. Acting on his statement, I secured X ray films of the right iliac fossæ of eight of my patients, they being those who were available, and at the same time those in whom the operation had been performed a sufficient time ago. Not one of them showed any calcification; and the most surprising thing about the series is that the patient whose glands had at operation been found to be partly hard and calcified, showed only a vague and diffuse shadow, and certainly no calcification as one is accustomed to seeing it. This film was taken eight years after operation. The full significance of this unexpected finding is difficult to estimate, but undoubtedly the calcification process had ceased, and a large percentage of the calcium already deposited must have been absorbed. Many factors in the study of these adenitis cases and one's knowledge of tuberculous infections in childhood, would incline one to accept a tuberculous infection as taking a large part in the pathology of these glands. It is certainly hard to disprove it. But in a

large number of cases, and in this series of mine as well, it is equally hard to prove it, and my own feeling is that it does not play the preponderating part which has been ascribed to it in the past.

The situation of the glands affected, the slight congestion often observed in the appendix and lower part of the ileum, and more particularly the complete relief of symptoms afforded by the removal of the appendix, would seem to make it evident that it is from this area that the glands primarily become infected. On the other hand, however, one has to think of how comparatively slightly these glands are affected in such gross infections as acute appendicitis and the comparatively slight participation of the mesoappendiceal glands as compared with those of the retroperitoneal ileo-caecal group. For these reasons some writers consider that the condition is the result of a generalized infection entering by the tonsils, and having a specific affinity for lymphoid tissue. Pribram[3] claims that he has by removing the tonsils brought cure to patients in whom abdominal pains have persisted after appendicectomy. The peculiarly limited situation of the glands involved seems to make that theory difficult to maintain. Schnitzler[4] explains the sequence of events on the basis of a delayed infection, which has subsided in the primary site and disappeared, leaving the glands infected secondarily. In this series I have tried to find some facts to support this view, which seems reasonable, but have failed. All one can say at present is that the explanation of the condition in terms of pathology, involves one in considerable difficulties, of which the solutions are not yet available.

CONCLUSION.

This paper may appear to be of purely academic interest, and of very little practical importance, seeing that when the differential diagnosis between appendicitis and adenitis is made, the treatment remains the same—the removal of the appendix. That is true; but none the less I think the subject is of sufficient merit to warrant discussion.

Summed up, the position is that this condition represents a definite clinical entity, and with care and close observation it is diagnosable in a reasonable proportion of cases. It is curable by removal of the appendix, but is likely to persist until this operation is performed. Clearer recognition and closer study of the condition when it is met with will undoubtedly lead to the solution of the pathological problems involved at some future time.

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Surgical Technique.

INTERNAL SHORTENING.

By F. A. MAGUIRE,
Sydney.

As a preliminary to the operation of internal shortening, the patient is put in the lithotomy position, a catheter is passed into the bladder and the pelvis is examined bimanually. An effort is made to determine whether the uterus is freely mobile or fixed. The uterus is curetted and the patient is placed in the Trendelenburg position.

Step I.—An incision is made in the mid-line above the pubes. The centre of the incision should be opposite the abdominal inguinal ring, which is midway between the anterior superior iliac spine and the top of the *symphysis pubis* (Figure I). In thin subjects the incision need not be more than 5·0 centimetres (two inches) in length. It is deepened down to the aponeurosis for the full length of the wound.

Step II.—The subcutaneous tissue is picked up on one side of the mid-line and dissected off the anterior surface of the sheath of the rectus with the edge of the scalpel. In many cases, once the dissection is started, the subcutaneous tissue can easily be stripped back with a piece of gauze placed over the end of the gloved finger. The dissection is carried out for about 5·0 centimetres (two inches) from the mid-line, that is, to the outer edge of the sheath of the rectus. It is not necessary to carry the dissection out for the full length of the original incision. It should be triangular, with the incision as the base of the triangle and the outermost point of the reflection as the apex. While this reflection is being carried out, several small veins and arteries which are perforating the aponeurosis to enter the fatty layer may be encountered. These should all be tied, as any oozing may give rise later to a hæmatoma under the flap. If the correct plane is struck in reflecting the fatty layer, it will be found that it is adherent to the anterior surface of the sheath of the rectus only by thin filmy areolar tissue. It is shown in Figure II as being reflected with the handle of the scalpel.

Step III.—The second step is repeated on the opposite side.

Step IV.—The sheath of the rectus is incised (Figure III). The margins of the *rectus abdominis* muscles are displayed and separated, the extraperitoneal tissue being exposed.

Step V.—The peritoneum is picked up and incised (Figure IV), the abdominal cavity being opened in the usual way. The peritoneum at the lower end of the incision may be anchored to the skin by a stitch.

Step VI.—The margins of the wound are well retracted by small retractors held by assistants. These are better than self-retaining retractors for this operation. If the muscles are thoroughly relaxed under the anæsthetic and the table is placed so that the patient is in the high Trendelenburg position, the intestines will fall back into the upper part of the abdomen and will leave the pelvis clear. If not, they should be lightly packed back with a large moist sponge.

Step VII.—The pelvic viscera are now inspected. Any adhesions that may exist between the tubes, ovaries, uterus or other viscera are freed. The operator makes sure at this stage that the tubes and ovaries are healthy.

Step VIII.—A suture is placed through the *fundus uteri* (see Figure V) to act as a tractor to lift up the uterus. Frequently it will be found that there are adhesions between the pelvic colon and the peritoneum in the region of the left round ligament. These need to be freed thoroughly, so that the pelvic colon falls back into its proper position in the back of the pelvis, otherwise on drawing up the round ligament in the process of shortening the bowel may be kinked. This is a definite danger when the operation is done as an external shortening and the abdomen is not opened.

Step IX.—It is immaterial whether the shortening is done on the left or the right side first. In this description the shortening is drawn and described as being done on the left side of the pelvis first. The round ligament is picked up between two pairs of Moynihan's bile-duct clips at a point about 3·75 centimetres

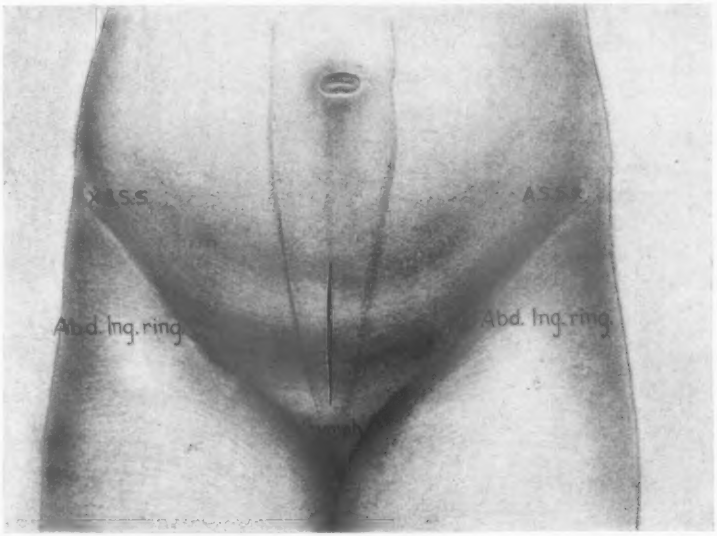
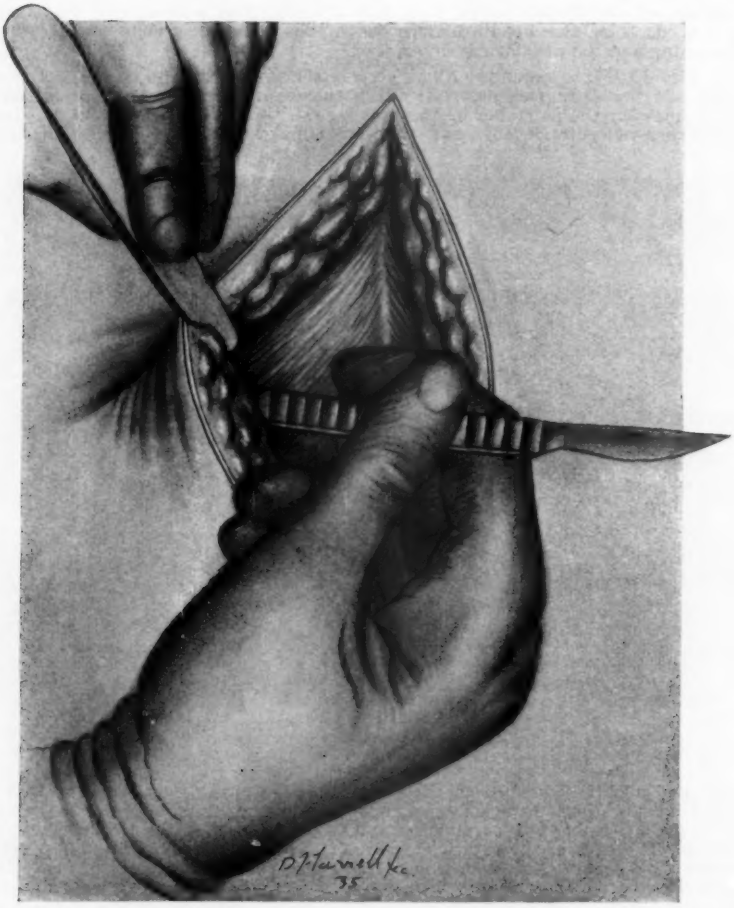


FIGURE I.

(one and a half inches) from the cornu of the uterus. Care is taken that the round ligament itself is picked up without any accompanying excess of peritoneum. A suture of number two chromicized gut is passed around the ligament and tied lightly. The ends of the suture (Figure VI) are then threaded through a special needle with a curve corresponding to the course of the round ligaments. These needles are after the style of MacEwen's needles, but are much larger and have a bigger curve. It is advisable to thread the suture through the eye of the needle from back to front, so that the operator is always sure on which side he inserts it, as at a later stage in the operation he must be quite sure to draw it through from the right side of the needle.

Step X.—The broad ligament is now placed in tension, and a small incision is made on the back of the broad ligament (Figure VII) with the point of the scalpel. This cut need not be more than eight millimetres (one-third of an inch) in length, and should be close up to the round ligament. The point of a pair of



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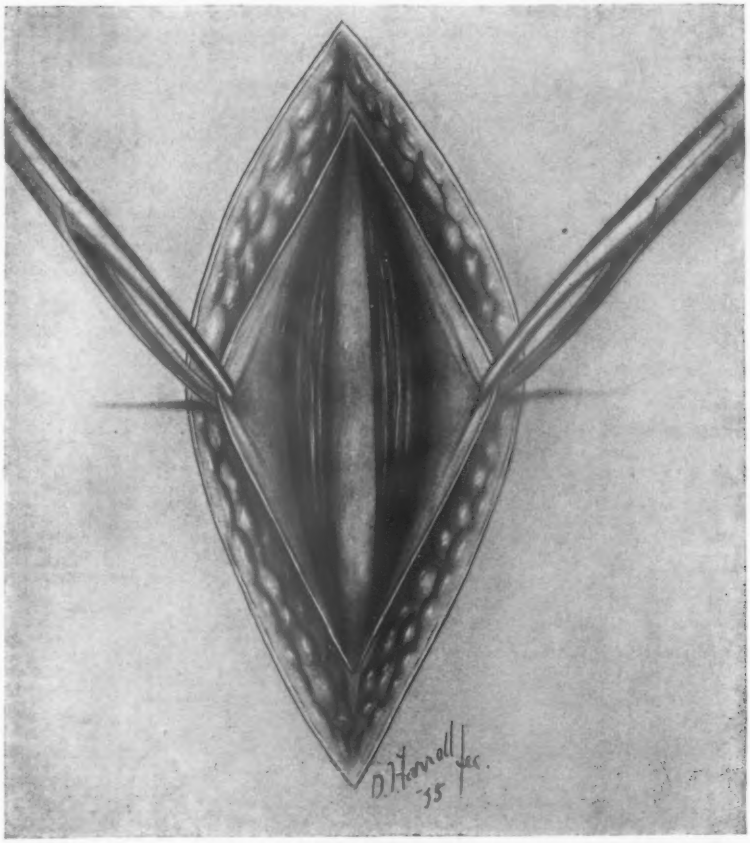

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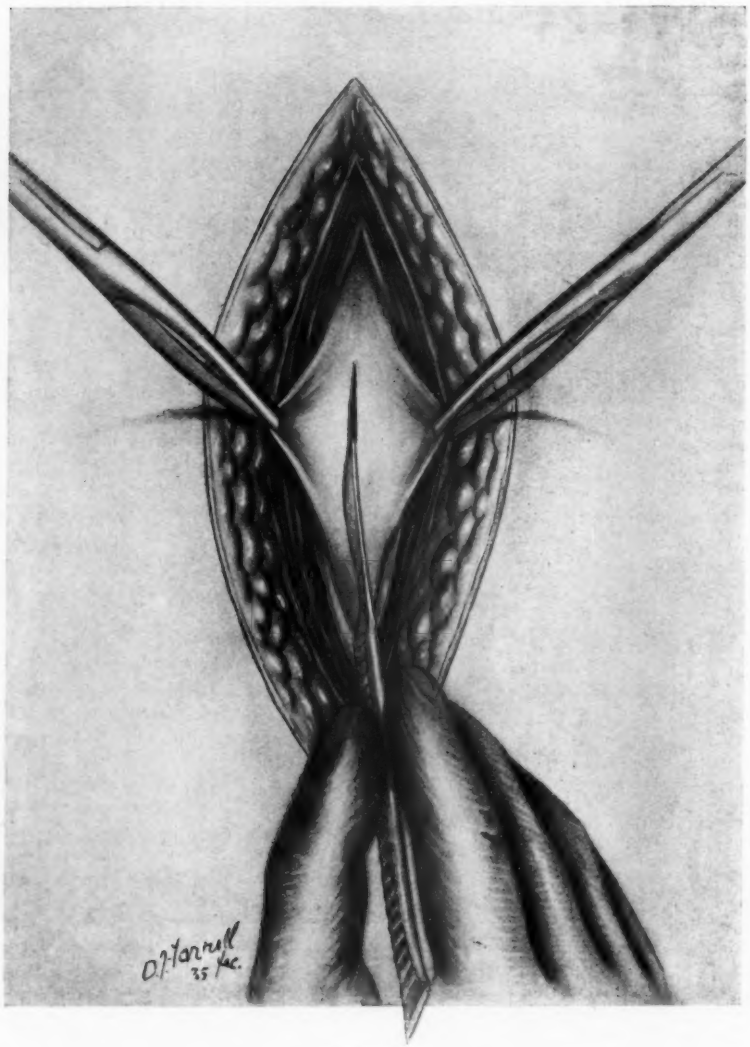

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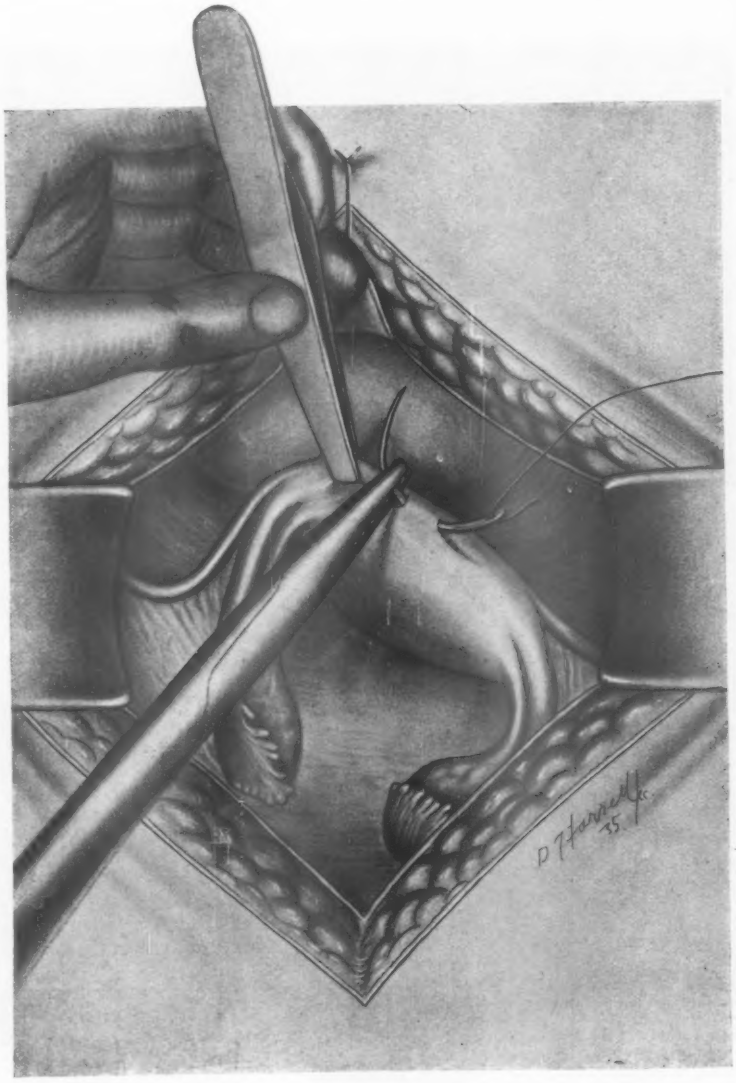

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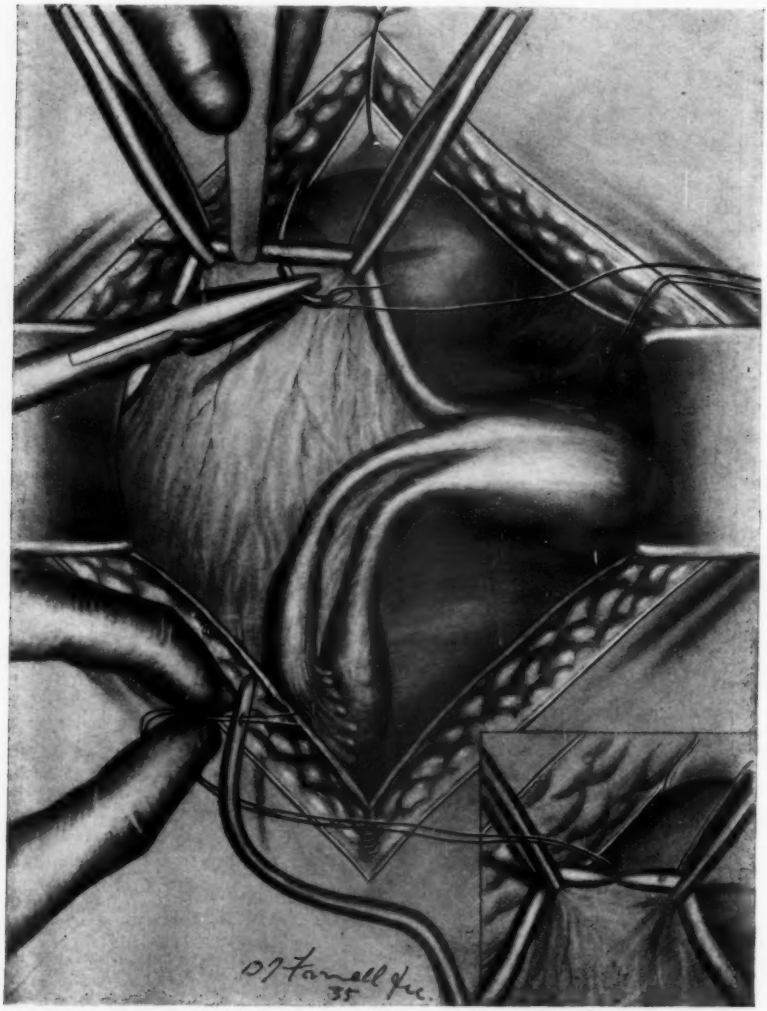

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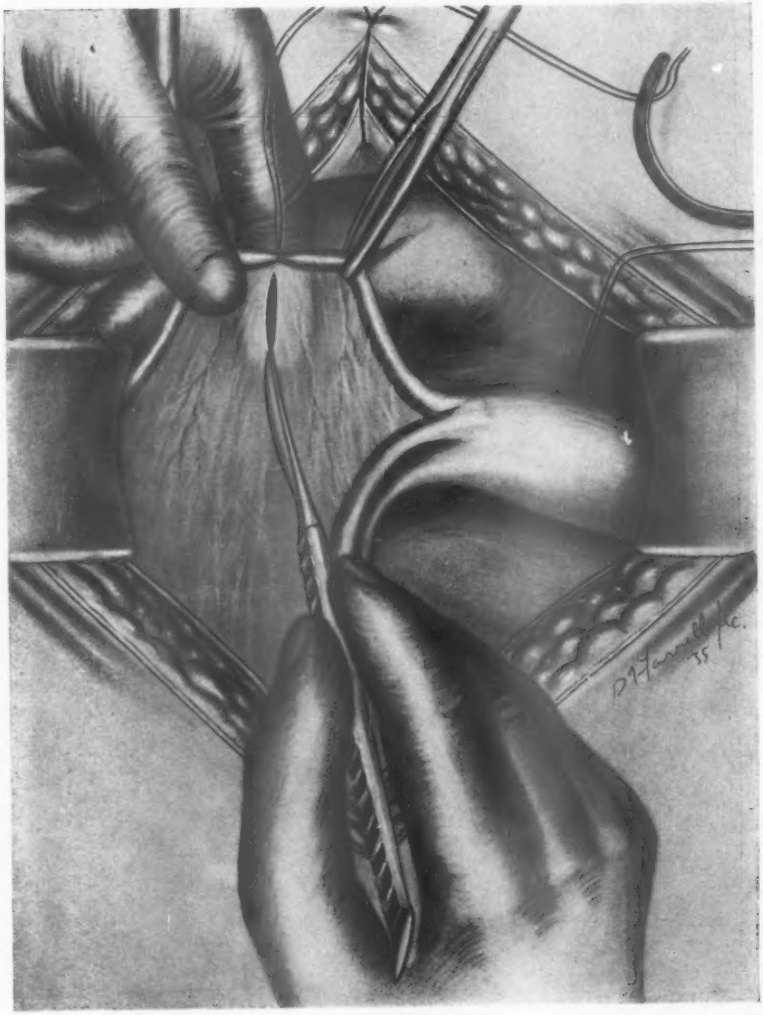

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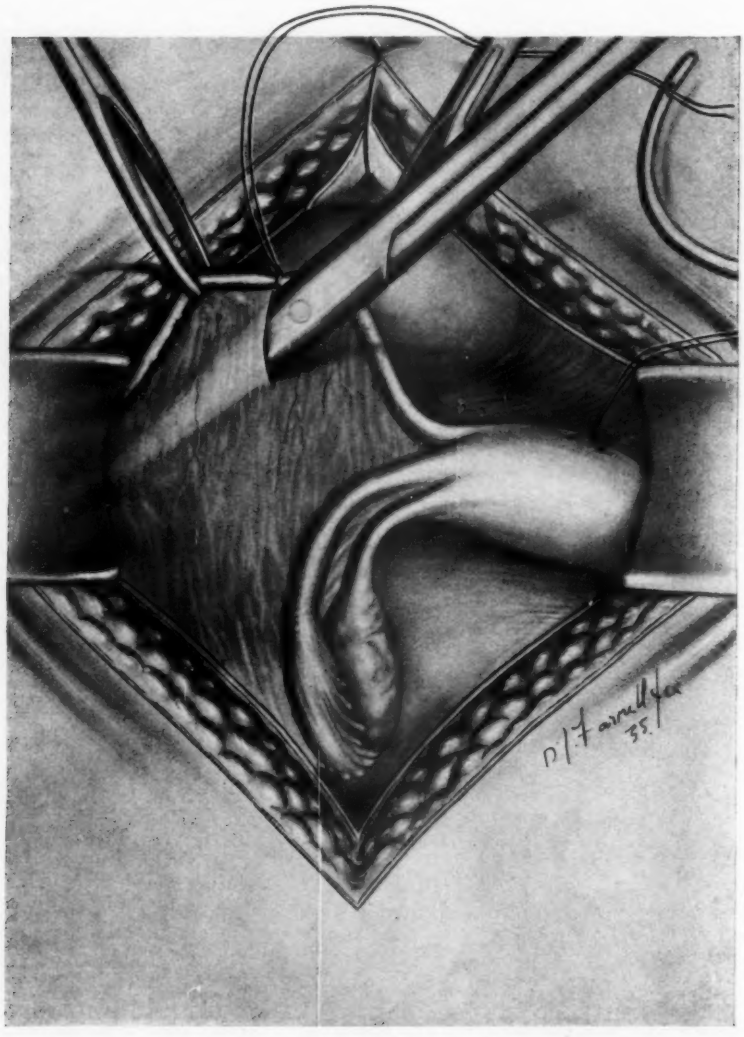

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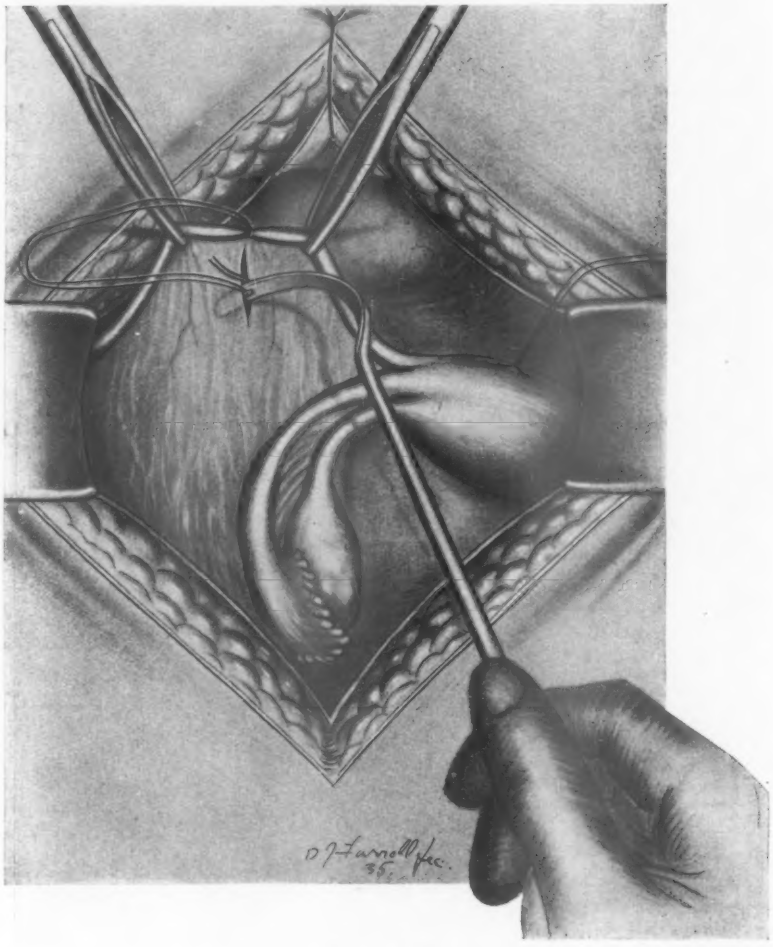

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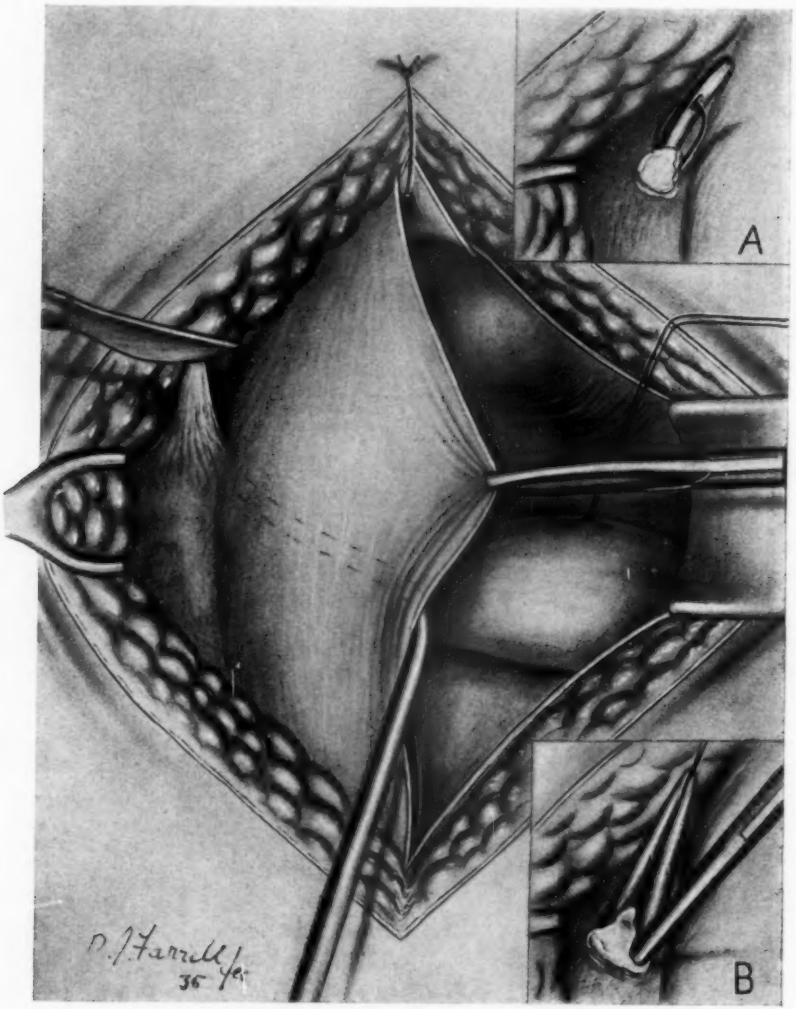

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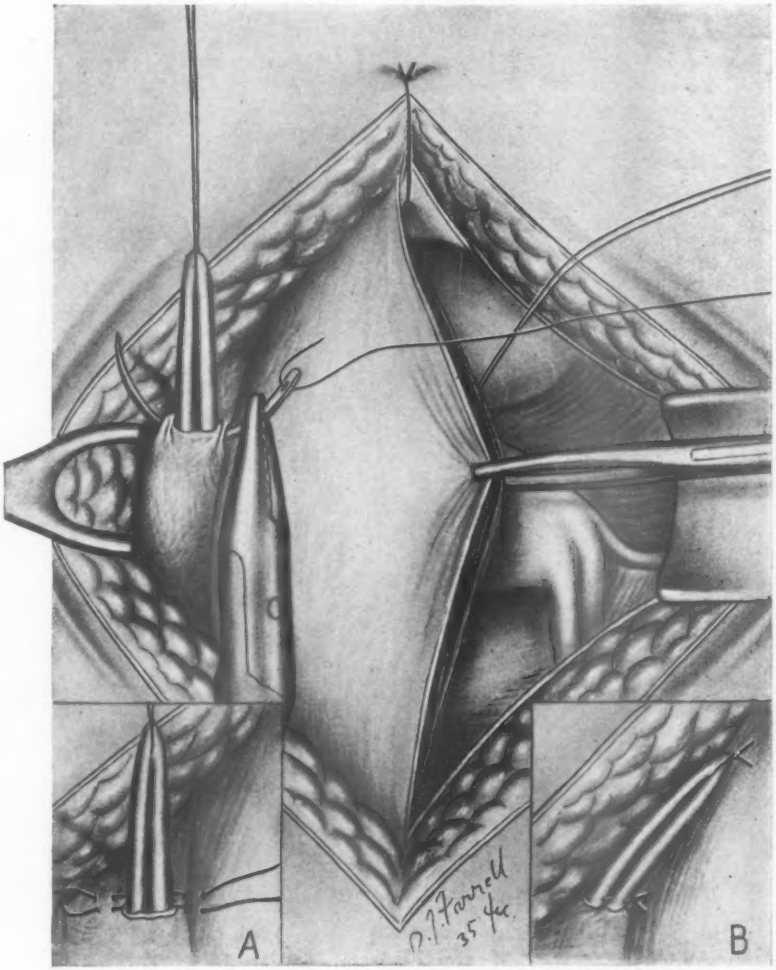

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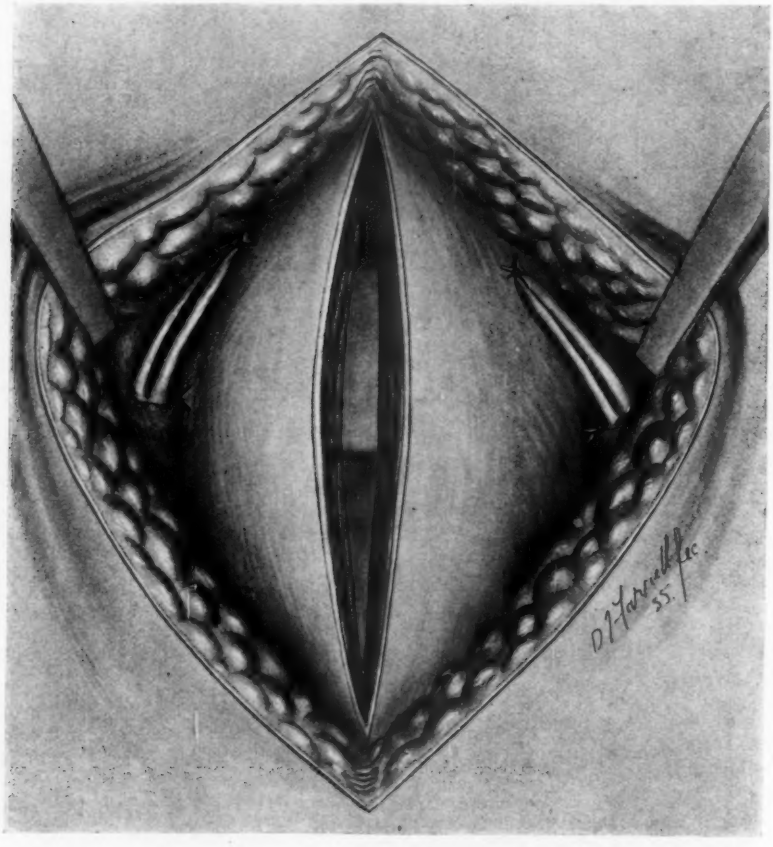

FIGURE XII.

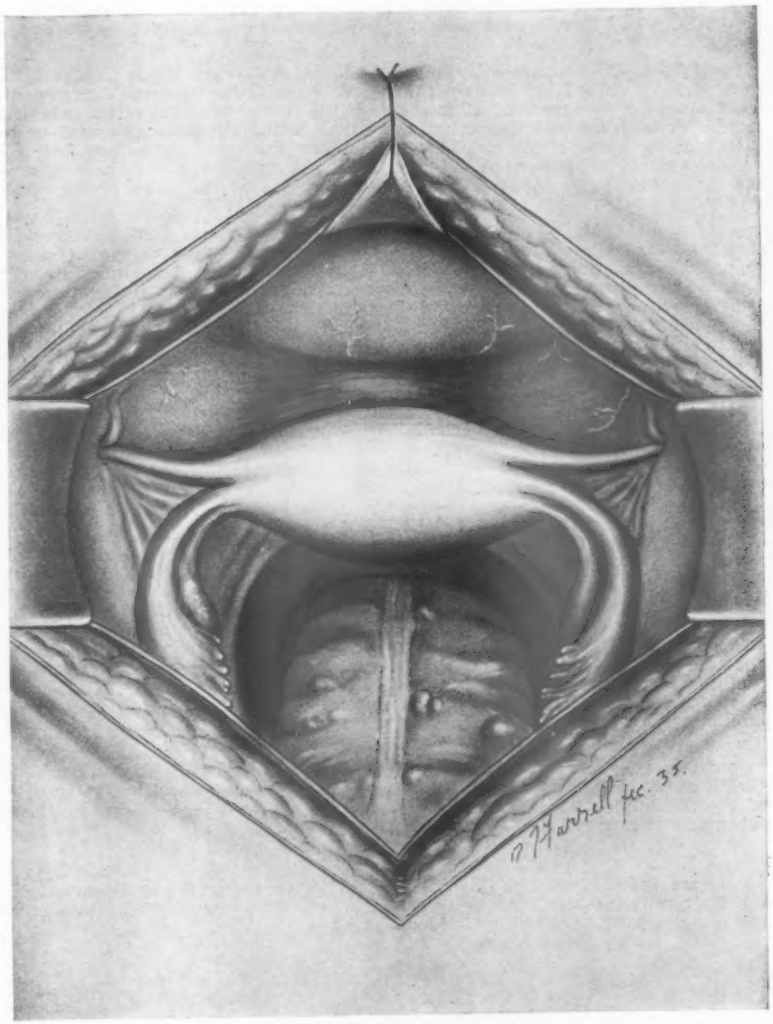

FIGURE XIII.

Mayo's curved scissors is now inserted and gently worked out between the layers of the broad ligament towards the brim of the pelvis (Figure VIII) in order to open up a little passage between the layers of the broad ligaments extra-peritoneally. If this is done carefully there need be no bleeding at all at this stage.

Step XI.—The carrier with the suture threaded through the eye (Figure IX) is now gently passed out between the layers of the broad ligament following the track made by the scissors. At the brim of the pelvis the point of the carrier is turned forward, the assistant at the same time lifting the abdominal wall on the same side by means of the small hand retractor. The point of the carrier is now pressed against the posterior surface of the anterior abdominal wall at a point on the same level as the abdominal inguinal ring, but at the outer border of the *rectus abdominis* muscle (Figure X).

Step XII.—An incision is made over the point where the aponeurosis is raised by the point of the carrier, and the carrier is slipped through (inset A, Figure X). It generally carries with it a prolongation of peritoneum. The ligature is unthreaded from the eye of the needle and the carrier is withdrawn. Gentle traction on the ligature now draws a double loop of round ligament through the abdominal wall (inset B, Figure X). The peritoneum that accompanies it is gently stripped back with a pair of forceps, and the double fold of ligament is drawn out as far as the surgeon considers necessary. It is usually necessary to draw out a loop about 2·5 centimetres (one inch) in length, which gives a total shortening of 5·0 centimetres (two inches) to the round ligament.

Step XIII.—A suture of fine chromicized gut is now passed through the anterior layer of the aponeurosis, piercing both parts of the round ligament. This is tied as a mattress suture (inset A, Figure XI). The suture at the end of the loop is now anchored to the aponeurosis on a line parallel with the inguinal ligament (inset B, Figure XI). Note that while the Steps XI and XII are being carried out an assistant retracts the fat and skin with a small hook retractor as shown in Figures X and XI.

Step XIV.—The preceding steps from IX to XIII are repeated on the opposite side. Both round ligaments have now been shortened to the requisite length. Figure XII shows the final stage of the shortening with the loop of round ligament lying on the anterior surface of the sheath of the rectus anchored at two points by fine chromicized sutures. Figure XIII shows the appearance inside the abdomen seen on retraction of the abdominal wall. There should be about 1·25 centimetres (half an inch) of round ligament clear from the cornu of the uterus to the abdominal wall, and the uterine tube should hang freely without any kinking.

Step XV.—The abdominal wall is closed in the usual way. It is advisable to put two deep silkworm gut sutures through the subcutaneous tissue and the sheath of the rectus in order to obliterate the dead space caused by the reflection of the subcutaneous tissues on either side. It is an added safeguard against subcutaneous hæmatoma.

Acknowledgement.

I am indebted to Mr. D. J. Farrell, artist to the Anatomy Department of the University, Sydney, for the drawings which have been done by him at the operating table from actual operations.

Case Reports.

A CASE OF NASO-PHARYNGEAL FIBROMA.

By Richard Flynn,
Sydney.

R.M., A MALE, aged twelve years, was first seen in consultation with the late Dr. Herbert Marks at Lewisham Hospital on June 9, 1927. He sought medical attention on account of blockage of the left nostril, facial deformity and loss of vision in the left eye. He had had no previous illnesses and had suffered no injuries.

FIGURE I. Showing tumour removed at operation.

His story was that during the last two months he had noticed blockage of the left nostril and failure of vision in the left eye.

On examination a large mass was seen protruding from and completely blocking the left nostril, and on inspection of his naso-pharynx a similar mass was seen; on palpation it was found to be fixed to the posterior wall of his naso-pharynx. It was also noted that the patient could not breathe at all through the left nostril.

An ophthalmic report by Dr. E. A. Brearley was that the visual acuity of the left eye was perception of light only; papillœdema of five diopters was present. The right eye was normal.

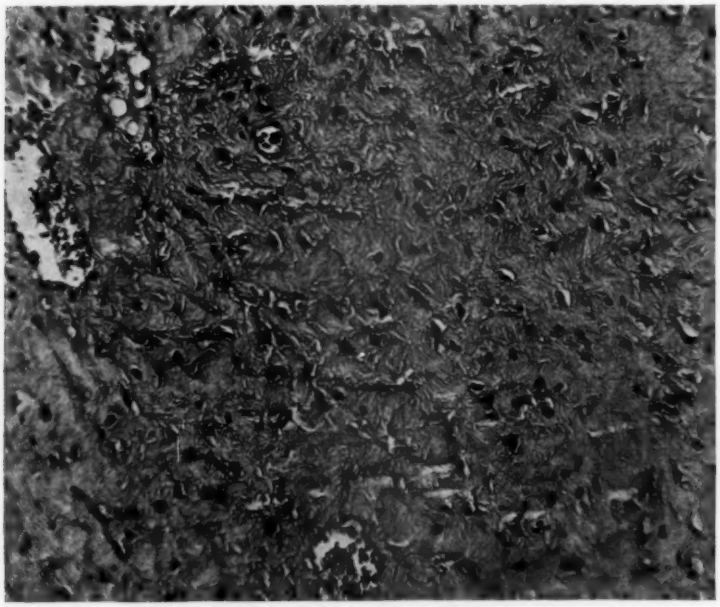

FIGURE II. Showing microscopic section of tumour.

Dr. Bede Harrison reported on a skiagram of his skull as follows: "Underdeveloped sphenoidal sinus, cloudiness of left maxillary and ethmoidal sinuses."

A biopsy was taken by Dr. Cyril Shearman, who reported that microscopically the section showed the structure of a fibromyxoma.

The patient was operated on in two stages. At the first stage the internal carotid artery was tied; during this procedure a gland overlying the internal jugular vein was removed. A Moure's incision was made on the left side, with the slight modification that the incision was continued up the columella, the left nasal aperture being thus kept intact. The nose was retracted to the right side and the anterior wall of the maxillary antrum along with the frontal process of the maxilla and the nasal bone were gouged away, the whole anterior surface of the tumour mass being thus exposed. The wound was closed.

A week later the wound was reopened and the entire tumour mass (Figure I) was removed. The mass marked O was drawn out of the orbital cavity. The large cavity resulting was tightly packed with vaseline gauze which was drawn out through the left nostril, and the wound was again closed.

Dr. Shearman reported that microscopic section showed the tumour to be a connective tissue growth showing fibrous and fibroblastic tissue with the same inflammatory change and also myxomatous degeneration (Figure II).

FIGURE III. Showing patient at the present time.

The condition must be regarded as a fibromyxosarcoma, but the sarcomatous element is not pronounced. Metastasis should not follow, but local recurrence is likely to follow removal. The gland removed from the neck showed inflammatory changes, but no new growth could be detected.

The patient was demonstrated at the recent meeting of the Royal Australasian College of Surgeons in Sydney (Figure III), and to date has shown no sign of recurrence. His vision in the left eye is now $^{5}/_{6}$.

A CASE OF VITAMIN DEFICIENCY SIMULATING GENITO-URINARY TUBERCULOSIS.

By J. W. S. LAIDLEY,
Sydney.

W.T., A MALE, aged twenty-four years, was referred to me complaining of frequency and scalding on micturition of eighteen months' duration and of hæmaturia in addition during the preceding six months. During this time he had lost about 12·6 kilograms (two stone) in weight. He had had no previous illnesses. During his school days he had been a noted athlete, securing colours for football and athletics. On leaving school he had become a school teacher, and for the last eighteen months, that is to say, for the period of his illness, he had been appointed to a town in western New South Wales.

On examination, his colour was poor, he had obviously lost a great deal of weight, his right knee and left ankle were swollen and painful on movement, and on palpation both joint capsules were distended with fluid. He was passing urine painfully every five minutes day and night.

A specimen of urine was subjected to Dr. Marjory Little, who reported the presence of much pus and blood. A culture was sterile, and a prolonged search failed to disclose any tubercle bacilli. On cystoscopy under low spinal anæsthesia, the bladder wall was found to be intensely inflamed. *Per rectum* the prostate and vesicles seemed to be normal, and there was no swelling or induration of testes or cords.

In hospital the patient displayed a voracious appetite for all kinds of food, especially for salads and green vegetables. In a week his joints had subsided, and his frequency of micturition and scalding were far less. A "Uroselectan B" examination revealed apparently normal renal pelves, and a second cystoscopy, to my amazement, showed an almost normal bladder wall, with a patchy and obviously resolving cystitis.

A week later he was normal in every way. He had put on 4·0 kilograms (nine pounds) in weight, his joints were normal, his micturition was normal and painless, and his urine was free from pus and blood.

Seen three months later, he had regained his old weight and was leading a normal life. On questioning him closely about his diet during his illness, I elicited the fact that during the eighteen months he was stationed in the west he had had green vegetables or fruit on only three occasions. Also, when the hæmaturia developed six months before he saw me, his local doctor denied him meat. On inquiring upon what he lived after this deprivation, I was informed that for the last six months he had had nothing to eat but condensed milk and biscuits.

The outstanding points about this case were the extreme severity of the symptoms, the profound prostration of the patient and the rapid and complete recovery. No therapy whatever was instituted, with the exception that fresh foods of all descriptions were made a feature of an unrestricted menu. I am strongly of the opinion that the patient's troubles were due to a severe grade of vitamin deficiency.

The Australian and New Zealand Journal of Surgery.

All articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Vol. VI. OCTOBER, 1936. No. 2.

THYREOTOXICOSIS.

All students of goitre in Australia and New Zealand are indebted to the British Medical Association for including the subject of thyreotoxicosis in the deliberations of the annual meeting at Melbourne in 1935. The discussion revealed the extremely sketchy nature of existing knowledge of the essential ætiology. Lord Horder, in his opening remarks, pointed out how controversial was the name, implying as it did that some toxic substance derived from the thyreoid was responsible for the syndrome. For this, the original theory, advanced by Mobius in 1886 and revived by Plummer in his dysthyreoidism conception of the origin of the symptoms, there is still no experimental proof. The glands from the most toxic cases of thyreotoxicosis are in the main experimentally inactive. Indeed, the biological activity of thyreoid glands removed from thyreotoxic patients varies inversely with the clinical evidences of toxicity.

The probability is, as Sir Thomas Dunhill implied, that the symptoms are due to increased and excessive amounts of normal

secretion, but until some laboratory test is available by which the thyroxin content of the blood can be measured, this view must remain hypothetical. The cause of this suggested excess of normal thyreoid secretion, whether due to stimuli intrinsic or extrinsic to the thyreoid gland, remains unknown. It was noteworthy that neither of the distinguished leaders of the discussion referred specifically to the much discussed "leader of the endocrine orchestra", the pituitary with its thyreotropic hormone, or to the suprarenal cortex, or to the ovary—all with possible relationships to thyreotoxicosis. In a clinical discussion it was appropriate to focus attention on the deranged function of the thyreoid gland, but there is much clinical experience and experimental work to suggest that the disordered thyreoid action may ultimately be traced in part at least to extra thyreoideal influences.

The essential thing to realize is that the name thyreotoxicosis is misleading, since it implies that the ætiology of the condition it denotes is definitely and exclusively thyreoideal.

No exception was taken to Dunhill's view that a sharp line of demarcation cannot be drawn between the various manifestations of thyreotoxicosis. No mention was made of the arbitrary division of thyreotoxic states into primary and secondary, with the manifold difficulties which surround this separation. No differentiation was attempted between "exophthalmic" and "toxic adenoma". If this unitarian view of thyreotoxicosis could be adopted by the Royal Australasian College of Surgeons, if only for statistical reasons, it would mark a great advance in the study of the subject in Australia and New Zealand. Sir Thomas Dunhill referred to the great increase in thyreotoxic mortalities in England and Wales in the last ten years, and speculated on the unrecorded morbidity. In Australia and New Zealand the mortalities from thyreotoxic states as recorded by the medical profession in death certificates are on record in the official vital statistics of the two countries. As in Great Britain, however, there is no complete record of the number of people who are admitted to hospital, private or public, for thyreotoxic states. If the College would adopt a uniform classification and would fix upon some simple method of recording and registration for all goitre cases, a distinct forward step would be taken. That there is an urgent need for some uniform classification is evident to anyone who has scrutinized the number of names which are attached to thyreotoxic states in different hospitals and even by different surgeons in the same hospital. A classification which might be considered is the one suggested by the National Conference of Nomenclature

of Disease, namely, non-toxic diffuse goitre, toxic diffuse goitre, non-toxic nodular goitre and toxic nodular goitre.

The British Medical Conference revealed that the incidence of thyreotoxicosis was notably higher in New Zealand, where endemic goitre is widespread, than in Australia, where endemic goitre is restricted in distribution. It was shown also that a high percentage of patients admitted to public hospitals with so-called simple goitre, up to 70% in the Dunedin Hospital, were, in some degree, showing one or other of the following symptoms: fatigue, palpitation, nervousness, tremor, irritability, mobile pulse, vasomotor disturbance, anomalies of carbohydrate metabolism, and yet as their basal metabolic rates were within normal range, such patients were regarded as suffering from simple goitre. The New Zealand results support the view that people with enlarged thyreoids are much more liable to show such minor evidence of thyreotoxicosis than are people with normal glands. The experience of the Royal Air Force that all candidates showing any degree of thyreoid enlargement have some degree of cardio-vascular inefficiency, is significant in this connexion. Such facts suggest strongly that increased attention to the importance of the prophylaxis of simple goitre would, at the same time, reduce the incidence of thyreotoxicosis.

The discussion left one with the impression that, in the present state of knowledge, reduction in the amount of functioning thyreoid tissue by surgical means offers the patient with thyreotoxicosis the best available treatment.

C. E. HERCUS.

Critical Reviews of Surgical Subjects.

ACTINOMYCOSIS OF THE HEAD AND NECK.

By S. S. GARDINER,
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It is now generally accepted that branched filamentous organisms which may be cultivated under anaerobic and/or aerobic conditions, are found in the oral cavity of apparently otherwise healthy individuals; and, though these organisms in this situation may not produce any clinical manifestations for long periods, one cannot overlook the fact that they may in certain circumstances become pathogenic.

This may happen as the result of trauma, either by the introduction of foreign bodies, by dental instrumentation or during mastication. Trauma reduces the bactericidal ability of the tissues so that organisms, already present in the oral cavity or deposited at the time of injury or carried from within the body to the injured part after the injury, are allowed to multiply vigorously. Once introduced, the infection may remain more or less localized or extend rapidly or gradually to adjacent tissues or distant regions. It is well to remember that these traumatic lesions may occasionally consist of only slight or inconspicuous abrasions.

Actinomycosis of the head and neck offers a broad field for profitable research to the general surgeon, and especially to the dental surgeon, as it is often associated with carious teeth or may develop after treatment of dental, periodontal or gingival infection, or may follow extraction of teeth. It may follow natural eruption of teeth or may possibly develop as a result of disturbances of nutrition or metabolism, particularly in children. The dental surgeon is often the first to see and treat this important and serious oral disease, the disease in its early stages being regarded as an ordinary dental affection. As illustrating how the alertness of a dental surgeon may lead to an early diagnosis and treatment, only one case need be mentioned.

Mills[1] reports a case in which a dental surgeon who was extracting some teeth, informed the patient of the serious trouble on his hard palate, of which, incidentally, he had no subjective symptoms. This early detection of the condition resulted in early treatment and apparent cure of a rare case of actinomycosis of the hard palate. Six months previously a splinter had injured the patient's hard palate, causing inconvenience for a few hours only.

The cases mentioned in this section demonstrate the necessity for careful X ray examination, with full appreciation of its limitations, in any case of disease in the maxillæ or mandible, as in this way small or even extensive areas of bone infection, sometimes not suspected, may be revealed and an isolated actinomycotic lesion may also be discovered. They also show the absolute necessity for a careful routine microscopic examination, and sometimes repeated examinations, of material obtained from slow healing mucosa, abscesses, discharging sinuses, areas of obstinate cellulitis *et cetera*, following tooth extraction, and also the necessity for an early consultation with a general surgeon when swellings in and around the jaws, face or neck follow interference with teeth, and, further, the importance of avoiding local injury to the tissues as much as possible.

It would be wise to consult frequently with a general surgeon, since the patient requires careful examination for extension of the infection as well as general treatment.

Each patient, too, should be warned to report to a dental surgeon or general surgeon if the mucosa is not healing readily or if a tumour appears immediately or even some months after dental treatment or extractions, as the patient frequently comes to have teeth treated or extracted by a particular dental surgeon, and immediately goes away a long distance to recuperate at some health resort. Unnecessary suffering is avoided by the intelligent observation and cooperation of the patient.

The dental surgeon should carefully listen to any symptoms of which the patient may complain, and in his reexamination of the patient should carefully examine not merely teeth which have been treated, or the cavities from which the teeth have been extracted, but the whole of the mucous membrane of the mouth, as well as the region of parotid, submaxillary and sublingual glands. He should also palpate the mandible for increased thickening, look for sinuses *et cetera*, being careful at the same time to observe the condition of the tissues of the neck, old scars, discoloration of skin *et cetera*.

If treatment has been prescribed, he should watch for signs and symptoms of the prescribed drug poisoning, and also be familiar with the reactions which may occur following vaccine, radium or X ray therapy.

The dental surgeon, too, realizing that the oral cavity contains numbers of potentially pathogenic organisms, the nefarious action of which is prevented by the soundness of the teeth and vitality and integrity of the mucosa, should carefully point out to all his patients the dangers of cavities in the teeth, dental fistulæ without obvious cavities *et cetera*. He should instruct his patients in oral hygiene, particularly patients suffering with actinomycosis in any part of the body, pointing out that a well kept mouth contains a less number of virulent bacteria than a badly kept one, as the oral hygiene of patients afflicted with this disease has been frequently sadly neglected. This need of dental education is well illustrated by a case of actinomycosis reported by Aguirre,[3] whose patient, aged thirty-two years, had not practised ordinary dental hygiene at any time. Furthermore, as actinomycosis is not peculiar to any particular age, the dental surgeon should instruct mothers to take their children regularly to see a dental surgeon from the time the child reaches two years of age, and should emphasize the necessity for periodic examination of the oral cavity, as well as regular toilet after meals; he should also insist that a physician be consulted if any disturbance of metabolism or nutrition is evident.

In 1925, Naeslund,[2] employing a medium containing sterilized saliva, isolated both aerobic and anaerobic types of actinomyces in material taken from dental scum, pulp and root canals of carious teeth, cysts, necrotic gums, saliva, mucous membrane of the mouth, tongue, tonsils and throat, and concluded that:

"Actinomyces may occur in the mouth under normal conditions, of which the more or less strictly anaerobic ones must be regarded as belonging to the normal mouth flora, while the luxuriantly growing aerobic types are presumably only chance organisms which have invaded the mouth from outside, and which have found suitable conditions for growth for a longer or shorter period in this situation."

He isolated strains of actinomyces which corresponded as closely as possible with the description of the organism isolated by Bostroem. He states that:

"Some of the preferably anaerobic actinomyces types isolated from the mouth under normal conditions are as nearly as possible identical culturally with the preferably anaerobic forms cultivated from cases of actinomycosis and which are commonly called Wolff-Israel's actinomyces."

In the following year, Naeslund[4] carried out investigations into the rôle played by various types of actinomyces and also leptothrix in the formation of tartar.

Bjerrum and Hansen[5] have made a systematic investigation for anaerobic actinomycetes in the pus obtained from gumboils and from carious teeth without abscess formation. Among one hundred and twelve samples of pus obtained from gumboils there were twenty-seven from which anaerobic actinomycetes were cultivated. The clinical course of these twenty-seven patients was practically

identical with that of the other cases; all were examined several months later and were found to have recovered after incision and dental extraction. Thirty teeth showed evidence of periodontitis, and among these there were twenty from which anaerobic actinomycetes were cultivated. All these patients recovered after extraction without any other treatment.

At a clinical meeting at which some of the cases tabulated at the end of this article were presented, the question was asked whether it would be wise to extract a tooth during the stage of any acute infection. That question has been well answered by Chompret and Dechaume,[6] who advise immediate extraction of carious teeth, when suppuration has passed through the periosteum and involved the subcutaneous and submucous cellular tissues, and in suppurative adenitis and osteomyelitis and maxillary sinusitis of dental origin.

They also describe the technique of extraction and after-treatment, emphasizing the need to insert the needle into healthy tissue, to sterilize the needle between each injection, and to avoid unnecessary trauma.

The possible disastrous effects of overlooked trauma to apparently healthy tissue are well illustrated by a case recorded by Stengel,[7] in which a girl, aged fifteen years, who had been in good health, consulted her dental surgeon. While filling one of the upper right molar teeth, the dental surgeon accidentally let the instrument slip, penetrating the cheek slightly. He caused a slight injury, from which there was a little bleeding, not sufficient, however, to prevent filling the tooth, but which was followed by an extensive actinomycotic lesion of the cheek.

It is interesting to note also that two cases of possible direct transmission of the disease from man to man may have resulted from wounds caused by infected teeth. In both cases the inference is that the organism of actinomycosis was present in the mouth of a person, who was not suffering from the clinical signs of the disease, and that the infective agent—virus, granule, portion of filament or whatever it may have been—was carried into the tissues of the victim as a result of injury caused by infected teeth. The infective agent possibly also may have been already present on the victim's skin.

Cope's[8] patient, during a fight with another man, three months previously, had knocked out his opponent by a hard blow on the upper jaw. His fist struck against his opponent's teeth, knocking them out, and caused a small superficial wound on the back of his hand. Inflammation ensued, and after an acute stage passed into an indolent condition. Later considerable infiltration developed in the first intermetacarpal space, and actinomyces granules were found in pus discharged from a sinus in that region.

McWilliams's[9] patient six months previously had cut the proximal knuckle of his third left finger on an adversary's tooth, and, though the wound healed in four days, a fusiform slightly bluish coloured swelling subsequently developed on the finger, which was amputated. Microscopic examination revealed the actinomycotic nature of the infection.

In two cases reported by Mattson[10] the patients developed actinomycosis on the buccal mucous membrane opposite a sharp carious tooth, which had caused an abrasion in this region.

In one of a series of cases reported by New and Figi[11] actinomycosis developed while the patient was being treated for pyorrhœa, and in another case a primary lesion in the tongue developed immediately after the tongue was bitten; in six other cases the disease immediately followed extraction of teeth.

Pallares and Medina[12] report a case which presented nodular lesions in the cervical region following stomatitis with painful irritation of the gums a year previously.

Forder,[13] at the Seventh Australian Dental Congress, reported a case in which the maxilla above the apex of the left lateral incisor was swollen, and multiple sinuses in that region were discharging watery pus that contained yellow actinomyces granules.

Harmer and Just[14] report a case in which a tooth was removed to relieve an abscess in the upper jaw. A subperiosteal abscess was opened, the pus of which contained actinomyces granules.

Cases of actinomycosis of the lungs following the accidental aspiration of a tooth or portion of a tooth into a bronchus have been reported by Israel and Warwick.[15]

New and Figi[11] have reported a case following removal of tonsils, and four other patients gave a history of acute sore throat directly preceding the onset of their infection.

Mattson[10] also reported a case which began as a unilateral sore throat and in which the patient subsequently developed an actinomycotic peritonsillar abscess on this side.

Vermes[16] has reported two cases following the removal of tonsils.

Hofhauser's[17] patient had suffered pain in the throat for a year, and during the previous six months had noticed and removed white specks from her tonsils, which had been painful for several weeks before operation; histological examination of sections of the tonsils removed at operation revealed typical actinomyces colonies.

Castellani[18] calls attention to tonsillo-actinomycosis. This is a condition, generally unilateral, in which an abscess slowly forms in the tonsil and peritonsillar tissues, and may be accompanied by pain and difficulty in swallowing. The abscess may open spontaneously through one or several openings and discharge pus containing granules; the abscess continues to discharge for an indefinite period, and the infection may spread to other parts.

Lord[19] found organisms having the morphology and staining reactions of actinomycetes in smear preparations and in serial sections from the contents of carious teeth, and in serial sections of yellowish, hard or soft material from the tonsillar crypts of patients without clinical symptoms of actinomycosis.

In a study of ten thousand pairs of tonsils, Wilkinson[20] found 1·77% to contain granules in the crypts, which resembled those found in actinomycosis.

In seven thousand tonsils, actinomyces colonies were found in six cases only by Coller and Adie.[21]

Davis,[22] on the other hand, after examining granules from hundreds of human tonsils, believes that, though the actinomyces-like organism in the granules found in the tonsillar crypts bears a striking similarity to the organism described by Wolff-Israel, the morphological as well as the cultural characters are distinctly different.

Source of Infection.

In the literature many cases have been cited which tend to support the theory of autogenous infection advocated by Wright,[23] supported by Colebrook[24] and Naeslund,[3] and originally suggested by Israel:

"That *Actinomyces bovis* is a normal inhabitant of the buccal cavity, and that the part played by foreign bodies so frequently found in actinomycotic lesions is not that of a carrier of the microorganism into the tissues from without as originally suggested by Bostroem, but that the foreign body, by its traumatic and irritative effect, furnishes a nidus in the tissues for actinomyces, which enters therein with the secretions from the buccal cavity, develops into characteristic colonies and produces a lesion which is called actinomycosis."

According to their view, the saprophytic or pathogenic character of actinomyces in the oral cavity depends upon the capacity for reaction of the neighbouring tissues, and this capacity for reaction determines either the development of an actinomycotic lesion or successful resistance to infection.

Although autogenous infection caused by injuries to the buccal mucosa and infection from carious teeth due to a pathogenic actinomyces, which has apparently been leading a saprophytic existence in the oral cavity, has been cited by numerous authors as the cause of many cases of actinomycosis, we must remember the possibility of the introduction of a similar or even the same strain of actinomyces from without.

In one patient in our series in which numerous actinomyces granules were found in a periapical abscess, the patient attributed his condition to the use three weeks previously of a new toothbrush with very stiff bristles. In a careful search of available literature only two cases of actinomycosis were found in which infection was considered by the patient to be due to or possibly associated with brushing the teeth, a procedure which probably traumatizes the gingival mucosa considerably. A few of the early writers were of the opinion that actinomycosis was transmitted directly to human beings from definitely infected cattle. In a few cases an actinomycotic lesion developed after an injury by an apparently healthy animal. More than one writer refers to indirect infection from animals, as the result of drinking fresh blood from infected cattle, or as a result of infected material being carried on the hands of employees at slaughter-yards, and also as a result of material dropped from an infected animal being carried by air, dust, water or by agency of flies to man. A few early authors mention the possibility of infection from drinking milk obtained from infected cows, and also the chance of infection from consumption of infected beef imperfectly cooked. Several of the early writers, however, and one recent writer,[25] concluded from their observations that the sole source of infection of both man and animals was from infected Gramineæ and from the residue *débris* and the dust emanating from it. Though the literature during the past twenty years has been enriched with numerous observations and case reports, no satisfactory explanation has as yet been given to show clearly the source of infection of actinomyces isolated under microaerophilic or anaerobic conditions from lesions in man, and the life history and habitat of this organism under natural conditions outside of the human body still remain a mystery.

One author[26] has reported a case which suggests the possibility of infection from man to man. A nurse attending a patient with cervico-facial actinomycosis, who had been operated on in all about one hundred times, developed a lesion in the cheek. From the records, it appears that she had symptoms before the actual admission of the patient to hospital. In a case reported by another author[27] the bride of a patient suffering from actinomycosis in the region of the mandible developed the disease in the same situation. Though the author originally assumed that the infection was due to kissing, another author states that he later changed his view. A few authors have referred to Falge's[28] report of three cases of actinomycosis which occurred in children at an orphan asylum, and in which the author suggested the infection was probably due to direct contagion. It should be noted, however, that a definite diagnosis of actinomycosis was made in one case only. Wagner[29] relates that one of the pupils of Sacchi, having opened an actinomycotic abscess, touched his nose before he had disinfected his hands. His nose became infected, and he succumbed in a short time to fulminant actinomycosis. Christ[30] suggests that people who dwell in the city may possibly become infected from street dust, particularly when the lesion is associated with trauma, the organism of actinomycosis entering the body at the time of injury. One of his patients, living under ideal hygienic conditions and having no carious teeth, as the result of a bicycle accident suffered bilateral fracture of the lower jaw and severe lacerations of the soft tissues, which were followed seven weeks later by an abscess at the site of injury in the left mandibular region, and five months later by an actinomycotic lesion in the same region.

In a case reported by Loxton[31] the patient sustained a scalp wound following a fall on a kerbstone and later developed an actinomycotic lesion in the same region.

Keynes's[32] patient developed an actinomycotic lesion in her cheek following a slight injury by a straw. A lymphatic metastasis developed later in one of the glands of the neck.

Downes[33] has reported a case of actinomycosis of the left parotid region in a boy, aged thirteen years, which apparently resulted from a kick in that region while the boy was playing footbali.

Bacteriological Examination.

No useful purpose would be served by presenting an epitomized description of the varied cultural, morphological and biochemical properties of actinomyces which have been isolated from actinomycotic lesions in the region of the head and neck. It would also be of little or no interest to attempt to make a *précis* of various experiments which have been conducted on the filtrability and cyclogenetic development of actinomyces. It would serve no better purpose to describe organisms isolated by a few authors from cases of "pseudo" actinomycosis which have been described in the region of the head and neck.

One author[34] has stated that actinomyces isolated from lesions during the life of the patient are usually anaerobic, while actinomyces isolated from lesions in the cadaver are generally aerobic.

Most authors, many of whom have not attempted to isolate the causal organism in the described lesion, simply state that actinomycosis caused by the microaerophilic actinomyces described by Wolff and Israel is the only true actinomycotic lesion, whereas in reality the true meaning of their assertion is that the microaerophilic actinomyces is the species most frequently isolated in cases of human actinomycosis.

Colonies of the organism are at times very hard to detect in actinomycotic lesions, but if repeated microscopic examinations are made, definite colonies are sooner or later detected. They have been found in all the cases tabulated at the end of this article.

Macroscopic colonies, granules, or *Drusen*, generally appear as mulberry-like bodies and vary in consistency with age; young granules are gelatinous or cheesy, old granules calcareous. Young granules have a silvery translucent appearance and later become greyish, yellowish-brown, yellowish-green, grass green, dark green, black, white, light yellow or golden yellow.

Large granules are often found to consist of daughter colonies growing round a primary or parent colony, and this accounts for the mulberry appearance of the granules.

The size of the colonies varies from a decimal of a millimetre to about two millimetres in diameter.

Microscopic examination of a crushed unstained granule shows a radiating felt-work of wavy filaments with true branches and with distensions or clubs arranged round the periphery. It was also observed that all filaments did not have a distension or club, and that sometimes they extended out beyond the periphery of clubs. When stained by the Gram method with a counter-stain of dilute carbol fuchsin, a granule is found to consist of numerous wavy filaments and fragments of filaments entangled and lying at all angles to one another with numerous true branches irregularly distributed along the length of the filaments. Some of the filaments have a terminal distension or club. A considerable variation in the manner in which the filaments and clubs retain the violet Gram stain is also noticed. In some the protoplasm is not uniformly stained, but shows light and dark violet stained areas, while in others the protoplasm shows a Gram-positive granular appearance.

The granular appearance varies, as the protoplasmic granules or coccus-like bodies in the filaments and clubs vary in size and number.

Often a Gram-positive granular filament extends into a large Gram-negative club. Gram-negative filaments are also present in a number of cases.

Preparations made from the granules in our cases were never in any degree acid-fast.

Old calcareous granules appear to be non-viable when transferred to suitable media for growth of the organism.

Successful cultivation of actinomyces from lesions in twelve patients suffering from actinomycosis observed in this district, with one exception, shows that they are definitely similar in their morphological and cultural characters, a description of which has been given in a previous article.[35]

Early in the course of this research a patient who had been suffering from cervico-facial actinomycosis and chronic nephritis died as a result of acute

bronchopneumonia. The cervico-facial lesion in this patient was not regarded by the bacteriologist who was at the hospital at that time as definitely actinomycotic, and for this reason has not been included among those tabulated at the end of this article. The culture of actinomyces which was isolated from the lesion, on account of its ability to grow readily at 22° C. under strict aerobic conditions and its thick layer of ectoplasm which swelled out into large distensions or clubs at the ends of many filaments, conformed to the characteristics of the non-pathogenic actinomyces often found in nature.

Six years ago another patient seen in consultation was considered clinically to be suffering from an inoperable carcinoma of the floor of the mouth. The morphological characteristics of the organism eventually isolated from the lesion which later involved the lower lip and both cervical regions, were similar to those of the actinomyces isolated from the previous case, except that the organism did not grow readily at 22° C.

On microscopic examination of a crushed and unstained "sulphur" granule removed from the lesion in each case, the granule was found to consist of a radiating felt-work of mycelial filaments showing true branches with terminal distensions or clubs. Other granules were crushed and stained by the Gram method with a counter-stain of dilute carbol fuchsin, and were found to consist of numerous filaments and parts of filaments entangled and lying at all angles to one another; the filaments showed numerous true branches, and also showed a thick layer of capsular material or ectoplasm which swelled out into large distensions or clubs at the ends of many filaments. Some filaments were Gram-positive and in others the bacterial protoplasm was stained by the counter-stain, that is, it was Gram-negative, while the coccus-like bodies or granules present in the filaments retained the Gram stain and were Gram-positive. Some of the filaments have Gram-positive terminal distensions, in others the clubs are Gram-negative.

Microscopic preparations made from the primary granules and later from cultures were non-acid fast.

In the case included in this series, some of the granular material removed from various areas in the lesion under local anæsthesia was inoculated on the surface of sloped agar, sloped glucose agar, sloped glycerine agar, inspissated serum and sloped testicular agar, and also in stab agar, stab glycerine agar and stab glucose agar, and, as one would expect, the cultures were badly contaminated with other organisms.

Therefore, some of the granular material was treated in the following manner:

1. Absolute alcohol, 1 minute; sterile distilled water, 3 changes; it was then crushed and the material was cultured.
2. Sterile formal saline (0·4% formalin), 10 minutes; sterile distilled water, 3 changes; it was then crushed and the material was cultured.

All culture tubes inoculated from the material so treated as well as those made from the original untreated material were incubated at 37° C. under aerobic, microaerophilic and anaerobic conditions, but no colonies of actinomyces were found even after several months' incubation.

Many attempts were made to isolate the causal organism, and eight months after the initial experiments were conducted, two colonies were observed with a hand lens on one tube of sloped agar and a single colony on another tube after seventeen days' incubation at 37° C. under aerobic conditions. Each colony penetrated the surface of the media so that one-quarter of the colony was above the surface, while three-quarters were embedded in the media. Each colony was about one millimetre in diameter and had an irregular edge, and with the exception of the centre which was brownish in colour, was greyish-white, and when touched with a platinum needle was hard and resistant to the touch. After some little trouble in getting rid of contaminations (mainly Gram-positive diplococci) by repeated subculturing, the organism was obtained in pure culture and grew readily aerobically on sloped agar, sloped testicular agar, inspissated serum and in broth at 37° C. It seems, however, to prefer inspissated serum, as on this medium it grows more rapidly and the colonies are slightly larger.

On rubbing the platinum loop over the surface of one of these colonies, and then emulsifying the material in a drop of water on a slide and staining by the Gram method with a counter-stain of dilute carbol fuchsin, and on microscopic examination the preparations are shown to be pure cultures of an organism having morphological characteristics similar to those of the organism in the primary granules.

The appearance of colonies on sloped glycerine agar and sloped testicular agar is similar to that described on sloped agar, though the colonies on the sloped testicular agar are somewhat larger in diameter.

Broth cultures show a whitish woolly growth like coarse wool in the bottom of the culture tubes after three days' incubation at 37° C., without, however, producing any turbidity of the medium.

The optimum temperature for growth is 37° C., though it grows slowly and poorly at 22° C.

Culture tubes which have been heavily seeded show a profuse growth of small discrete colonies, while those lightly seeded show fewer but larger discrete colonies; the colonies are never in any case confluent.

This organism will not grow under microaerophilic or anaerobic conditions, though it may remain viable, as the following experiments show.

Carbon Dioxide Experiment.

Eight tubes of sloped agar inoculated from an inspissated serum culture were divided into two groups of four each. Two of the tubes in each group were placed under carbon dioxide (Rockwell's method), and not even after four weeks' incubation at 37° C. was there any growth in the carbon dioxide tubes.

The second group gave the same result, though the tubes were left standing at room temperature for eighteen hours prior to incubation at 37° C.

After incubation for twenty-eight days the surface of each of the carbon dioxide tubes was washed with 0·5 cubic centimetre of sterile broth, and this was subcultured on to the surface of a tube of sloped agar and incubated at 37° C. under aerobic conditions; growth occurred in all the tubes.

Subcultures were also made on sloped agar from the aerobic sloped agar tubes and incubated at 37° C. under aerobic conditions, and growth occurred in all the tubes.

Cultures made in shake agar, shake glucose agar and shake glycerine agar after three days' incubation at 37° C. show at the top of the media, extending from the surface downward for a distance of 15 millimetres, a zone of profuse growth; below this well defined zone there is absolutely no growth.

The colonies appear as very small greyish-white irregular masses.

Cultures made in stab agar, stab glucose agar and stab glycerine agar, after three days' incubation at 37° C. show a growth which tapers off towards the bottom of the line of the stab, and which extends downward from the surface of the media for a distance of 25 millimetres; below this absolutely no growth takes place.

The growth consists of greyish-white nodular colonies along the line of stab.

Inoculation Experiments.

In the available literature, with the possible exception of five instances, it is still very questionable whether a pathological process has been induced in ordinary laboratory animals which actually corresponds to the actinomycotic lesions found in man. Although Naeslund[(28)] has recently reported a successful inoculation experiment in a heifer eight months old, experimental investigation by others supports the contention that the possibility of producing progressive actinomycotic lesions in laboratory animals similar to those observed in man is remote. We, ourselves, found that when ordinary laboratory animals were inoculated with subcultures of the third generation, the experiments failed to produce progressive lesions when the animals were killed at intervals of from six weeks to six months. Experiments with both aerobic and microaerophilic actinomyces were conducted

on guinea-pigs, which showed that the organism was excreted from the blood stream into the bile and urine; cultures of the organism were obtained from these sources.

Fragments of filaments and clubs were seen within the leucocytes on microscopic examination of the urine and bile of inoculated animals.

Cervico-Facial Actinomycosis.

Cervico-facial actinomycosis has been found in individuals of all ages. Figi and Cutts[27] have reported a case in a child, aged two years. The same authors have observed that a history of trauma is associated with the development of actinomycosis more often in children than in adults, and also that a history of dental extractions and penetration of the oral cavity with particles of grain or grass is conspicuously absent in children.

A history of a previous acute attack or of repeated attacks of acute or subacute inflammation, tumour or sinus formation in the same or neighbouring regions over a short or long period, which abated with or without treatment, may be obtained. In adult males the skin over the affected area may be scantily bearded or entirely depilated. The skin may not be involved or its altered appearance may be the first sign of the infection to attract the attention of the patient. It may become infiltrated or diminished in thickness and perforated by one or many, short or long, straight or tortuous, and sometimes communicating, sinuses with thick or thin, and sometimes undermined edges; the sinuses may discharge continuously or intermittently, and sometimes only on the application of pressure, large or small amounts of serum or pus, varying in consistency and colour; the pus may contain few or numerous granules of varying colour, size, shape and consistency. A few or many, short or long, straight or tortuous, narrow or wide, continuous or interrupted, puckerings or wrinkles may be present in the surrounding or distant skin.

Induration of the skin and of the subcutaneous tissue may extend far beyond the actually infected area. The local lesion varies in size and character, may develop rapidly or be insidious in its onset; it may be unilateral or bilateral, single or multiple, and may involve a limited or extensive area and cause a small or large, hard or fluctuant tumour; it may take the form of an inconspicuous ulcer or form a "veritable cavity" in the side of the face; it may resemble a small boil or large carbuncle. Inflammatory œdema of the muscles of mastication may produce trismus, which may be the earliest symptom of the onset of the disease. Pain may be entirely absent or may precede or accompany the appearance of the lesion or may appear after medical or surgical treatment, X ray or radium therapy; the pain varies in severity and character, and may be continuous or intermittent and more or less strictly limited to the location of the lesion, or it may extend over almost the whole of the patient's head. Pain may or may not be aggravated by palpation or muscular movement. Fever also may be entirely absent, intermittent or continuous, and may precede, accompany or follow signs of infection of the cervico-facial region. The progress of infection varies according to the type and virulence of the causal organism, the site of invasion and the presence or absence of superadded infection, and the presence or absence of metastases due to the organism of actinomycosis or superadded infection. In some cases the initial signs and symptoms of infection in the cervico-facial region may be attributed to a carious tooth or an injured area in the oro-pharyngeal cavity. The gingival mucosa may become painful and swollen, and eventually one or many teeth showing little or no evidence of caries may become loosened or even fall out; and if the lesion extends and involves the floor of the mouth in addition to the cervico-facial region, it may also interfere with deglutition and cause progressive increasing dysphagia. An isolated cervico-facial lesion may extend upward and attack the brain, backward to the vertebræ, or downward to the mediastinum; or it may cause metastases by way of the blood stream in near or distant regions, depending upon the localization, direction of spread and virulence of the infection. Isolated superficial lesions in the cervico-facial region frequently extend locally for a long time, hæmatogenous dissemination of the

infection being generally absent. When, however, isolated infection is situated in one of the internal organs, hæmatogenous invasion generally occurs early.

Apparently isolated lesions in the cervico-facial region may be the first and only outward manifestation of pyæmic infection. The patient may die within a few days of the appearance of the lesion, and an incorrect certificate of death may be issued unless autopsy is performed. In passing, it may be mentioned that in one case of pyæmic actinomycosis[38] in which an organism, cultivated under aerobic conditions, was isolated from the blood during the life of the patient, a perforation of the cartilaginous portion of the nasal septum was the only manifestation of pyæmic infection in the head and neck region. It is essential to remember that one or more diseases may coexist in the same patient and possibly in the same lesion; cancer and actinomycosis have been known to occur together in the same lesion in a patient suffering from syphilis.

Actinomycosis of the Lips.

At least four definite cases of isolated actinomycosis in the region of the lips have been reported. In Harger's[39] case a mass which was not adherent to mucous membrane or skin was removed from the substance of the lower lip. Merian's[40] patient, who was subject to epileptic seizures, noticed an itchy red spot about the size of a hemp seed above the left angle of her mouth; a mass about the size of a pea was removed three months later. Von Baracz's[41] patient observed a small "wart" on his lip, and injured it a number of times with his teeth. Clinically the tumour resembled a retention cyst; and a foreign body, which was probably a piece of straw, was found in the mass. The patient remembered having his mouth filled with pieces of straw at his employment two weeks previously. Von Baracz quotes Fantino and Grillo's case in which an actinomycotic abscess was said to have formed in an operation scar on the lip twenty days after an operation was performed for recurrent carcinoma. Koch's[42] patient had a small nodule in her lower lip removed, and six months later, apparently from the same place, a hard infiltrated area below the margin of the lip was removed, which contained actinomyces granules. In another article published by Koch[43] mention is made of a case of actinomycosis of the lower lip reported by Esmarch; no details of the case are given.

Actinomycosis of the Jaws.

Actinomycosis may be found in the jaws as a simple localized lesion in dental cysts and abscesses, as chronic periodonitis, as recurrent alveolar abscesses and occasionally as an epulis. A few authors have observed that numerous and large metastatic lesions may develop in various regions from a small and circumscribed apparently primary focus in and around the jaws. Other organisms may help to cause this extensive spread of infection. Infection has been known to develop in cases in which the jaw has been fractured.[44] Perimaxillary types are generally the result of extension of the infection from the soft tissues surrounding the maxilla. Actinomycotic osteomyelitis of the jaws, at least in its early stages, does not differ clinically from osteomyelitis produced by other organisms. X ray examination of an advanced lesion either in the upper[45] or in the lower jaw[46] gives no indication of the actinomycotic nature of infection in this situation. In at least one case of rarefying actinomycotic osteomyelitis of the lower jaw, reported by Chompret and Dechaume,[47] there was no involvement of the soft parts or no evidence of secondary infection. As Chompret and Dechaume[48] have observed, "too often the descriptions given are those of an advanced stage of the disease—a pathology of the dying". Trismus may not be very manifest, and may even be absent if the surrounding tissues are not affected. When infection of the soft parts does exist, the overlying skin does not always have the violet-red appearance described in many textbooks, neither is it necessarily perforated with multiple fistulæ. In one case reported by Dechaume[49] "masses of mycelial filaments" were found in microscopic sections of a sequestrum removed at operation. Central actinomycosis of the maxilla or mandible is comparatively rare, although three cases have been reported by Klingsbigl[50] and two cases

by Hashimoto,[31] and may be found in two forms—central caries and central neoplasm. In a case reported by Cope[48] egg-shell crackling was elicited on applying firm pressure on a large cystic tumour of the mandible. In one case recently reported by Meyer-Borstel[73] the organism may have invaded the bone through the mucous membrane. The lesion was situated in the ramus of the mandible, where normally no tooth is present. Hæmatogenous invasion, which is known definitely to occur in bones in other situations, cannot be definitely excluded in this case. The maxillary antrum may be infected with actinomycosis and the actinomycotic infection may sometimes also be associated with carcinoma.

Actinomycosis of the Tongue.

The tongue may be involved by an isolated infection or as a result of the extension of infection from the surrounding tissues and also by hæmatogenous invasion. In 1921 New and Figi[52] published three cases of primary actinomycosis of the tongue and tabulated thirty-five cases from the literature. In available literature up to 1934 additional cases of isolated actinomycosis of the tongue have been published by McWeeney,[54] Kazda,[58] Bonnet,[59] von Hedry,[57] Figi,[60] Grupen,[59] Erb,[60] Barlow,[61] Hlaváček,[62] Banister,[63] Cannon,[64] Wanaker,[65] Eiken,[66] Snoke,[67] Mitchell,[68] Zimmermann,[69] McQuay,[70] Venetianer[71] (who mentions Funfack, Lawen and Lumniczers's cases), Cameron,[72] Moulonguet,[73] and Mazzini and Palazzo.[74] Kellock[75] mentions an additional case. Edye[76] has mentioned one case observed in New South Wales, and Cade,[77] in an article published last June, mentions that he had seen a dozen cases in the previous ten years. An isolated primary lesion may be painless and cause no discomfort apart from its presence. The infection may be acute or insidious in onset. The mucosa may have a normal appearance or be discoloured and elevated above the surface or margins of the tongue. The lesion may appear as an abscess, ulcer, cyst, sinus, a small or large tumour, or rarely as a diffuse indurated lesion with multiple sinuses. In some cases there is a definite history of trauma or of excessive smoking. A foreign body has been found in eleven cases, sometimes only after microscopic examination, and in a few cases apparently entered the tongue without the patient's knowledge. Sometimes the lesion recurs locally, and in Mayer's[78] case the patient died as a result of the extension of the lesion. In Kochel's[79] case the lesion was at first diagnosed as sarcoma, and later reexamination of sections of the lesion revealed the presence of actinomyces colonies. The lesion was associated with carcinoma in three cases, and in one case the patient also suffered from syphilis.

Actinomycosis of the Thyreoid Gland.

Actinomycosis of the thyreoid gland may be the result of the direct extension of a cervico-facial, laryngeal, pleuro-pulmonary or vertebral infection, or rarely the result of hæmatogenous spread of infection. In generalized pyæmic actinomycosis, the lungs, brain, kidneys, liver and spleen are most commonly involved; the myocardium, voluntary muscles, bones, meninges, skin and intestinal tract are less frequently affected, and the pancreas, testicle and thyreoid gland are most rarely the site of infection. In generalized pyæmic actinomycosis there may be small abscesses in and about the thyreoid gland as in the case reported by Shapiro,[80] or solitary and few in number and relatively large in size as in the case described by Fellinger and Salzer,[81] in which an abscess the size of a walnut was found in the right lobe and an abscess the size of a pea in the left lobe of the gland.

In the available literature isolated infection in the thyreoid gland itself has been reported only on two occasions, although a few authors have reported an isolated actinomycotic abscess in the region of the thyreoid gland which was not due to extension of a laryngeal or cervical focus of infection; in one case[82] the condition was alleged to be due to friction and pressure by a collar stud. In one case[83] the disease was regarded as a case of primary actinomycosis of the skin. The patient, aged twenty-five years, suffered from myxœdema. A tumour extended from the cricoid cartilage to the chin. In places it presented a bluish-red

appearance and simulated scrofuloderma. The tumour became fluctuant in places and, on incision of the mass, granules were noticed in the pus, the actinomycotic nature of which was verified by microscopic examination. The affected skin was removed, and the thyreoid gland was found to be partly destroyed, only about one-half of the gland remaining. The affected tissues were curetted. Not only was a local lesion thus cured, but the symptoms of myxœdema were greatly improved. One year later the patient was still apparently well. The second case[70] occurred also in a female, who presented "a condition of actinomycosis of the thyreoid gland, the diagnosis being confirmed by biopsy". Four years later the patient was apparently well. An interesting personal case of isolated actinomycosis of the thyreoid gland was operated upon in 1920.

Mrs. W., aged thirty-six years, since the birth of a child eleven years previously had been suffering from glycosuria and a cystic goitre, which was confined to the right lobe. As the tumour was becoming more difficult to conceal, she consulted a herbalist, who painted the tumour with some "black stuff" each morning for ten days. Three days later the skin over the centre of the tumour became purplish-red and adherent to the tumour, and severe pain interfered with her sleep and necessitated the use of sedatives. Two days later she was advised to have the "abscess" in her neck incised. Granules, subsequently found to be actinomyces granules, were found in the faintly yellow gelatinous material which escaped on incision of the mass. An elliptical incision was then made around the discoloured area of the skin which covered the mass, and the remaining portion of the right lobe and also portion of the left lobe were removed with difficulty, and the wound was allowed to remain wide open. The cavity was irrigated with 10% hydrogen peroxide each day for eleven days, and was exposed daily to the action of sunlight. The glycosuria disappeared on the sixth post-operative day, and the wound was apparently healed in six weeks. Potassium iodide was administered three times a day on the second post-operative day in doses of 0·6 gramme (ten grains), and the dose was gradually increased to 2·4 grammes (forty grains) three times a day on the twelfth day, and was continued for four months, when the patient refused to take any further treatment. Apart from one attack of bronchitis she was apparently well, until her death occurred, two years later, following an attack of influenzal pneumonia. No other organism was found on examination of direct smears and in cultures of the gelatinous material in the tumour. On section, the cystic tumour, which measured nine centimetres in diameter, was found to have a distinct capsule, which was about one millimetre in thickness. The lateral and posterior portions of the remaining portion of the right lobe of the gland were apparently healthy, and the capsule of the tumour showed myxo-fibromatous tissue on microscopic examination, and was highly vascularized by endothelial channels. As the patient admitted that she had frequently injured the skin overlying the tumour with a pin which she inserted into a scarf that she used to conceal the tumour, one naturally concludes that the infection was associated with trauma; but in view of the gelatinous consistency of certain isolated cystic actinomycotic tumours found in the region of the third ventricle of the brain and in other regions, infection as the result of hæmatogenous invasion cannot be disregarded.

Possibly careful microscopic examination of sections of all degenerated cystic goitres removed at operation or autopsy will increase the number of case reports of isolated actinomycosis of the thyreoid gland.

Actinomycosis of the Salivary Glands.

Söderlund, in 1909, reported two cases of actinomycosis of the submaxillary and sublingual glands respectively, in one of which a foreign body was present. These two cases were also reported with four others in another article published in 1913.[84] A foreign vegetable body was present in four of these cases. The following year he reviewed thirty-one cases, and wrote a detailed description of actinomycosis of the salivary glands. This series included the first definitely proved case of primary and ductogenic actinomycosis of the parotid gland. On the inner surface of the cheek in this case a hard nodule, about the size of a walnut, could be

palpated and continued in the form of a strand to the parotid gland; apparently it was produced by a thickened Stenson's duct with infiltration of the surrounding tissues. Pressure upon the strand resulted in the discharge of pus containing typical actinomyces granules from the orifice of Stenson's duct, which opened on the summit of the above-mentioned nodule. In 1921, Söderlund[85] published four new cases which he had treated, in which, in his opinion, the organism invaded the affected parts along the excretory ducts of the salivary glands, as in his other cases. In the second case in this series a rounded tumour, about the size of a hen's egg, was palpated in the right submaxillary region; it was of firm consistency and had no evidence of fluctuation. The skin over the tumour was not reddened nor adherent to the mass, which was not attached to the mandible. When pressure was applied to the mass, odourless pus which contained no visible granules exuded from the orifice of Wharton's duct. Microscopic examination of the pus revealed the presence of characteristic actinomyces colonies. In the fourth case, which was not associated with the presence of a foreign body, and which was similar to one reported in a child three years old, an abscess had developed in the cheek.

In explaining the mode of invasion in this case and in other cases in which the lesion was found in the cheek, Söderlund calls attention to the anatomy of Stenson's duct, and particularly to the sudden change of direction in its course; this, in his opinion, seems to explain why it is highly probable that actinomycotic lesions develop in the cheek in preference to other regions in the vicinity, especially when the lesion is associated with the presence of a foreign body introduced from without or when it results from the formation of a calculus in the duct. Precisely in front of the place where actinomycotic lesions of the cheek frequently appear, that is, close to the anterior edge of the masseter, the direction of the parotid duct suddenly changes. This duct, after appearing from under cover of the anterior border of the gland, runs forward across the outer surface of masseter muscle, then, turning suddenly inwards at the anterior border of the muscle and at right angles to its former course, it runs obliquely through the buccinator fascia and muscle to open into the vestibule of the mouth on the apex of a papilla opposite the second molar tooth of the maxilla.

Another feasible explanation of the location of isolated actinomycosis of the cheek in this region, particularly in those cases in which there is no history of trauma and in which no foreign body is present and the teeth appear to be perfect clinically and radiographically, is furnished by the cases of actinomycomata reported by Sebba[86] and Dobrzaniecki[87] in recent years. In these cases infection probably invaded the cheek by way of the lymphatic vessels. It seems possible that isolated actinomycotic lesions which have been definitely known to occur in an isolated fashion in glands situated under the sterno-mastoid muscle and in other glands in the neck, may also occur in the buccinator lymphatic glands. During the past thirty years at least, surgeons have always been careful to examine these glands when a carcinomatous lesion has been present in the eyelid, nose, upper lip, upper jaw, parotid region and tonsil, and since the organism of actinomycosis is now known to spread by lymphogenous invasion, it appears at least possible that actinomycosis of the skin and subcutaneous tissues in this region may be due to extension of the infection from the regional lymphatic glands. Söderlund,[85] in his eighth report on actinomycotic lesions in the salivary glands, suggests that sialolithiasis, Kuttner's tumours and primary actinomycosis of the salivary glands are ætiologically and pathogenetically the same affection, and are the result of ductogenic infection of the glands by the invasion of actinomyces from the oral cavity into the excretory ducts of the salivary glands. Naeslund[88] concluded from his observations and experiments that sialoliths were produced by actinomyces and not by other bacteria, and were possibly aided by physical and biochemical conditions in the salivary glands, which conditions are distinctly favourable for the development of concretions. Cazzamali[89] has recently reported three cases which support the contentions of Naeslund and Söderlund. It is well to remember that enlargement of glands in the submaxillary region may be associated with infection in other regions, as

in Montgomery's[91] case. In this instance the patient, aged twelve years, sought advice on account of bilateral enlargement of glands in the submaxillary region. The glandular enlargement subsided after treatment of a small yellow actinomycotic nodule in the soft palate and another in the pharyngeal wall. Other writers have observed that ductogenic infection with actinomyces does not always lead to the formation of calculi or definite actinomycotic lesions in the glands.

One interesting personal case of a calculus in the submaxillary gland of a female patient, aged fifty years, has been seen recently. In this case an organism has been isolated in pure culture under microaerophilic conditions; it was the only organism associated with the presence of the calculus. No evidence of actinomycosis, that is, pathological changes induced by the colonization of the organism in the substance of the gland, was found in numerous sections of the gland examined microscopically. As we have not observed a case of isolated actinomycosis of the salivary glands, and as most of the foreign literature is available in abstract form only, a review of the literature on isolated actinomycosis of the salivary glands has not been attempted. A sufficient number of original articles dealing with infection in the salivary glands has been examined to show that isolated actinomycosis in these regions may pursue an acute, subacute or chronic course, and that the pathological changes which result from the entrance of the organism cannot be differentiated clinically from pathological lesions produced by almost any other organism.

Actinomycosis of the Larynx.

The literature on internal and external laryngeal actinomycosis was reviewed in 1933 by Beysiegel.[92] In this summary the cases recorded by Arrowsmith,[93] Behr[94] and Eckert[95] have not been included. Another case was recorded by Riecke[96] in 1934. In available literature only two cases of apparently isolated intralaryngeal actinomycosis have been recorded. Actinomycosis in this region is generally the result of extension of infection from the oro-pharyngeal cavity or from the cervico-facial region, or is associated with pulmonary actinomycosis. The patient may complain only of slight hoarseness and an irritating cough. There may be slight or severe dyspnœa on exertion and slight or marked difficulty in deglutition. In one case dyspnœa was so severe that tracheotomy was necessary to save the life of the patient.

Perichondritis, œdema and infiltration of the epiglottis and unilateral or bilateral, localized or diffuse œdema and infiltration of the larynx may be present. The larynx may be displaced to one side of the neck and sometimes the larynx is rotated slightly on its vertical axis.

Superficial single or multiple abscesses and fistulæ discharging pus, which in some cases contains macroscopic granules, may be present. Two patients were also suffering from tuberculosis of the lungs.

Actinomycosis of the Ear.

Most of the reported cases of actinomycosis of the ear are the result of extension of infection from neighbouring tissues. Beysiegel[92] collected fifteen cases in the literature up to 1933. Very few cases of isolated infection of the ear have been reported. In Pochon's[97] case the infection commenced in an aperture pierced in the lobe for ear-rings. Pinaroli's[98] patient suddenly experienced pain in his ear, and ten days later observed exuding from his ear pus which contained "little grains of sand". Beysiegel's[92] patient developed a superficial abscess behind the ear after a furuncle had formed and had spontaneously ruptured into the external auditory canal. The pain at the onset of the infection was attributed to an unerupted third molar tooth, which was extracted. Zaufal's[99] patient suffered from an apparently primary lesion in the middle ear, and died from hæmorrhage due to an erosion of the vertebral artery. Zanni's[100] patient also appeared to have a primary infection of the middle ear, and eventually developed a retro-auricular abscess. Telling and Gruner[101] mention a case which was apparently successfully treated and in which the disease affected the external and middle ear and the mastoid cells. Figi[102] has mentioned that up to 1929 he had

seen at the Mayo Clinic only one instance of proved involvement of the mastoid in over two hundred and fifty cases involving the head and neck. The portal of entry in cases of actinomycosis in the middle ear is often very obscure. There was no definite indication of the point of invasion in six cases, in six the infection was the result of the extension of infection from a lesion in the parotid region, in five cases the infection apparently entered by way of the Eustachian tube, and two patients appeared to have been infected through the external auditory canal. Isolated lesions as a rule are acute in onset and the clinical features are not characteristic.

Actinomycosis of the Lachrymal Ducts.

Great difficulty was found in obtaining sufficient case reports in order to make anything like a complete review of the literature on actinomycosis in the lachrymal ducts. Most of my records are merely abstracts of articles. A number of cases have been reported from Russia, where the disease appears to be prevalent, and, judging from the abstracts of articles by Dutch authors and the complete article by Koster,[103] who saw three cases within a comparatively short space of time, the disease must be prevalent in Holland. It must also be prevalent in France according to abstracts available and the statement made by Rocher,[104] who, at a clinical meeting in 1914, stated that the case presented was exactly like others which he had already shown at clinical demonstrations.

One observation which impressed me early in the study was the large number of cases which occurred in females. Lagrange[105] furnishes an explanation of this discrepancy. He remarked that in nineteen out of twenty cases the patient was a woman; and he explained that women were more often occupied than men in the light duties of weeding the fields and removing hay *et cetera*, duties which placed them in contact with the plants harbouring actinomyces. Scant mention is made of the size and extent of the lesions; slight swelling was noted in a few cases. In Terson's (senior)[106] patient, aged seventy-two years, the tumour was the size of a small nut. A few authors who diagnose their patients to be suffering from actinomycosis of the lachrymal ducts, merely state that a "streptothrix was present". Some omit to mention whether cultivation of the organism was attempted and, if so, whether it was successful or otherwise. A number of authors, following Ginzburg's[107] advice, agree that a pure culture undoubtedly has a definite significance for the study of the biology and morphology of the organism in this situation, a microscopic examination being all that is necessary for a definite diagnosis of actinomycosis.

Sushkin[108] and two other authors who described lesions of the superior lachrymal duct were not certain whether their cases were typical actinomycosis or pseudoactinomycosis. Koch[109] obtained a pure culture of an organism resembling that described by Bostroem from a patient suffering from a polypus in the upper canaliculus. Wegner[110] mentions Stock's article, published in 1925, which contained the records of twenty cases in which cultures of actinomyces were successfully obtained. In many cases the organism required strictly anaerobic conditions, in some the organism was a faculatative anaerobe, and in others strictly aerobic conditions were necessary for its growth.

Although most writers consider that transport by air of contaminated dust to the conjunctival surface and from this situation into the lachrymal duct is the most likely mode of infection, Cartais[111] refers to a reported case in which water was regarded as the vehicle of transport of the infection, and Robert[112] also recognizes the possibility of water being the vehicle for conveying infection to the conjunctiva. It now appears to be generally agreed that the organism of actinomycosis is a normal inhabitant of the conjunctiva of otherwise apparently healthy people and possibly also of the lachrymal passages, and is not a very offensive guest, causing annoyance only by its presence and slow progressive colonization in certain cases. I am unable to find in available literature one case of actinomycosis of the lachrymal ducts in which the disease has recurred after operation, nor have I found any record in which the organism *per se* has caused the death of a patient. Actinomycosis in this location does not appear

to have the same tendency to relatively slow or less commonly rapid extension of spread of the infection as occurs when the disease affects most other situations in the head and neck. In fact it seems peculiarly strange that this disease, which is generally so serious, assumes such a benign character when the lachrymal passages are involved, particularly when the infection has been present apparently for years.

Many and varied hypotheses have been advanced to explain this peculiar phenomenon. One author maintains that the organism, having found its way into a lachrymal duct, encounters an unusual condition, in that the duct is constantly irrigated by "antiseptic" tears and, further, that the irritation produced by the presence of the organism induces hypersecretion of mucus which protects the mucous membrane against invasion by the organism. Another author maintains that tears constitute an excellent medium for the cultivation of actinomyces. An attenuated virulence of the organism as a result of contact with air and the destructive influence of other organisms found in symbiosis with actinomyces have been alleged to be the cause of the benign character of the lesion. This is demonstrated, one author says, by the fact that actinomyces fail to grow in cultures in which other organisms of secondary infection are developing. These circumstances lower the vitality and virulence of the organism, and may explain the infrequent colonization of the organism in the lachrymal ducts. The essential conditions for the evolution of the organism are certainly not definitely known; possibly slight trauma may be a requisite condition.

Three cases of actinomycosis of the lachrymal ducts have been observed in this district, the organism in one of which was successfully cultivated under microaerophilic conditions. Four macroscopic granules were obtained from the lachrymal duct of one patient, three from another patient, and in one patient who had signs of infection for only six weeks a single granule was removed from the duct. In all cases the lower lachrymal duct was involved. One patient whose symptoms had been present for four months, definitely stated that he had for the previous six months been very careful to dry his eyes thoroughly, "particularly in the corners", as a relative had developed trouble in his eye which eventually necessitated its removal.

Intraorbital Actinomycosis.

Cases of apparently isolated intraorbital actinomycosis are very rare; even cases of definite secondary infection due to extension from a focus of infection in the eyelids, in adjacent sinuses and the oro-pharyngeal cavity are rare, only eighteen cases being reported in available literature. The case reported by Bartoli,[112] in which the orbit was exenterated and showed "obvious traces of infection", has not been included in this number.

Although proptosis and swelling of the eyelids have been reported among the signs of some cases of actinomycosis involving the head and neck, it seems possible that the orbital cavity may have been involved in some of these cases, although no mention of the result of the examination of the orbital cavity at autopsy has been recorded. A few cases of intraorbital and cerebral invasion from an apparently primary focus of infection in or near the eyelids have been reported, one of which was alleged to be due to trauma.

In most cases the infection invaded the orbit as a result of extension of an actinomycotic lesion of dental origin, or from a lesion in adjacent sinuses, the pharynx and cervico-facial region. In at least two cases in which a dental focus of infection was regarded as a primary source of infection, intraorbital invasion was apparently due to direct extension of the infection through the soft tissues of the face along the lateral surface of the maxilla into the anterior portion of the orbit. In other cases the infection involved the masseter muscle, extended to the infratemporal fossa, and then entered the posterior portion of the orbit through the inferior orbital fissure. In two cases, infection of the orbit apparently resulted from extension of infection in the maxillary antrum on the same side, and in one the infection apparently invaded the orbit from the ethmoid cells. In one patient, whose teeth were apparently healthy, a large retropharyngeal abscess

was present. The infection in this case probably originated in the mucous membrane of the pharynx or tonsil, extended upwards to the spheno-maxillary fossa and entered the orbit through the inferior orbital fissure.

Beigelman[114] observed from his study of the literature that in spite of the long duration of actinomycotic infection in the temporal and infratemporal fossæ, no case of intraorbital infection had been reported to be due to direct spread of the infection through the lateral wall of the orbital cavity.

In Müller's[115] case the whole of the osseous wall of the orbital cavity was thickened by inflammatory new formation of bone, which reduced the size of the orbital cavity considerably. In his case a Brun's resection of the malar bone and lateral orbital wall was performed. Instead of having the normal thickness of three to five millimetres, the lateral wall, which was perforated by the infection, was two centimetres thick in some places. As the actinomycotic lesion was particularly extensive in the posterior portion of the orbit, and infection could readily pass through the inferior orbital fissure, Müller regarded the perforation of the lateral wall as a late result of the spread of the infection, either from within outward or in the opposite direction.

The intraorbital lesion may be extensive and involve the ocular muscles, nerves, vessels and even the bulb; or it may be of slight extent, as in Ransom's[116] case, in which the greater part of the orbital cavity was found to be healthy and separated from an actinomycotic sinus along its outer wall by a layer of firm fibrous tissue.

In the reports of five autopsies it was shown that the actinomycotic process invaded the meninges and brain, not only from the orbit, but also through the foramina and bones at the base of the skull.

In cases in which data are given, the duration of life from the first manifest symptoms of intraorbital infection to death varied from ten weeks to several months.

The signs and symptoms of isolated intraorbital actinomycosis in the early stages of its development are not characteristic. Pain, local or referred, sometimes unaccompanied by the usual signs and symptoms of inflammation, disturbances of vision, disturbance of motility of the eye, swelling of the lids, proptosis with displacement of the eye in the affected orbit and increase in the bulk of retrobulbar tissue frequently render it impossible to differentiate clinically between lesions caused by the organism of actinomycosis and those produced by other organisms, pseudo-tumours and tumours of the orbit.

The clinical diagnosis is easy in cases of secondary actinomycosis; these might be termed the typical and bad cases, in which definite colonies of the organism have been found in a focus of infection in adjacent sinuses, the oral cavity or pharynx; these foci of infection have preceded or accompanied the signs and symptoms of intraorbital infection, and in these circumstances one or more sinuses discharging pus containing granules are present in the neighbourhood of the orbit.

The prognosis is unfavourable, as death usually results from extension of infection by the organism of actinomycosis or by superadded infection, which develops in association with actinomycosis of the orbital cavity; of the extension of infection there may be no clinical manifestations during the life of the patient.

The surgical treatment in most cases has been limited to incision of abscesses, curettage of sinuses and insertion of a drain into the cavity of the orbit. The orbit was exenterated in the cases reported by Leahey and Dalmeijer.[117]

Actinomycosis of the Central Nervous System.

The youngest patient in available literature up to 1934 with definite cerebral actinomycosis was aged eleven years, the oldest seventy years. It is impossible to determine the frequency with which the brain is involved in cases of actinomycosis, as in some cases in which cerebral symptoms have been present, the child or adult has been taken home from hospital and no autopsy has been permitted; in other cases in which permission to open the cranial cavity has apparently not been refused, the autopsy surgeon has neglected to make, or has

failed to report the result of, this examination, or has simply stated that the brain was affected. In other cases the report of the microscopic examination of the specimen removed at operation or autopsy has been incomplete or omitted altogether. In some cases of metastatic infection a primary focus of infection has not been carefully sought. In 1882, Ponfick[118] reported two cases of cerebral actinomycosis; in one case the infection in the neck and prevertebral tissues became generalized and actinomycotic abscesses developed in the left cerebral hemisphere, pericardium, lungs and liver. The following year Zemann[119] drew attention to the autopsy report of a case of peritonitis in which actinomyces colonies were found in a right pyosalpinx and superadded infection only in abscesses which developed in the brain, lungs and liver. Keller's[120] case appears to be the first case in which the infection was diagnosed and operated upon during the life of the patient.

There is still great diversity of opinion as to whether isolated cerebral actinomycosis is truly "primary" or whether it should be regarded as metastatic infection. As the diagnosis of isolated actinomycosis of the brain has always been made at autopsy, and as secondary metastatic infection is sometimes recognized only after death, the presence of a single isolated abscess in the brain containing macroscopic granules would always stimulate a search for a primary focus of infection. Unless one is familiar with the macroscopic appearance of certain unusual isolated cerebral actinomycotic lesions, an isolated lesion may pass unrecognized, and the opportunity of discovering a near or distant focus of infection, which may be very small and also not macroscopically typical of actinomycosis, may be lost, particularly in those cases in which the real primary lesion has produced so few symptoms and signs during the life of the patient that it has been unrecognized by the clinician and possibly also unobserved by both the patient and doctor. It is essentially necessary to be familiar with the macroscopic appearance of these unusual isolated cerebral manifestations of actinomycosis, when the patient is definitely known to be suffering from coexistent tuberculosis, malignant disease, syphilis or *diabetes insipidus*, or when the patient has been suffering from symptoms suggesting a cerebral tumour, and a cyst or tumour resembling a true or false cystic tumour due to other pathological conditions in the brain is found at autopsy.

In available literature nine cases are recorded in which isolated cerebral actinomycosis has been found at autopsy, and the presence of definite actinomyces colonies has been demonstrated by microscopic examination. It must be pointed out, however, that the actinomycotic lesions have not always been the actual cause of the death of the patient. These cases have been reported by Bollinger, Buday, Enriquez and Sicard, Russkich and Krilova, Sternberg, Hallervorden, Kroner, Melnikowa-Rasvedenkowa and Zajewloschin. Zajewloschin's[121] case is the only case of isolated pituitary infection recorded in available literature. The patient, aged twenty-four years, had symptoms of *diabetes insipidus* for seven months, and symptoms of meningitis became manifest two months before death. The symptoms and autopsy examination suggested that the infection originated in the hypophysis and spread to the neighbouring meninges of the base of the brain. A pathological process at the apex of the right lung showed no evidence of actinomycotic infection. On section of the enlarged gland a narrow strip of apparently normal tissue was visible in the anterior portion of the periphery of the mass, the remaining portion being composed of caseous nodules. The condition found at autopsy suggested caseous tuberculosis of the hypophysis and tuberculous basal meningitis. The correct diagnosis was made only after microscopic examination of the mass.

In the case reported by Enriquez and Sicard[122] the patient, aged forty-three years, was admitted to hospital suffering from a cerebral tumour. The skull was trephined and a meningocele developed in the cranial wound, which was followed by suppuration in that region. Typical actinomyces colonies were found on microscopic examination of the pus discharged from the summit of the meningocele.

Russkich and Krilowa's[123] patient, aged thirty-three years, suffered from epigastric pain, flatulence, attacks of vomiting for two weeks prior to admission

to hospital and headache of two years' duration. The patient had been operated upon for a condition diagnosed as tumour of the left orbit one year previously, but no evidence of tumour was found at operation. Autopsy revealed a tumour, the size of a walnut, of firm consistency and irregular surface, in the left *crus cerebri* and adherent to the posterior wall of the orbital cavity. Histological examination of the tumour revealed the structure of a typical actinomycoma. Although no other focus of infection was found at autopsy, the authors suggest that the eyelids were possibly the site of invasion of the organism.

Bollinger's[124] patient, a female, aged twenty-six years, presented symptoms of a cerebral tumour twelve months before her death. At autopsy both lateral ventricles were enormously distended and an oval cystic tumour with smooth surface, pale greyish-yellow in colour and the size of a large hazel-nut, was situated between the anterior pillars of the fornix and attached to its lower surface by a pedicle. Its lateral surface was attached by bands to the median plexus of veins and was partially surrounded by the laminæ of the *septum pellucidum*. Its lower surface rested upon the anterior commissure of the third ventricle. The tumour resembled an ovarian cystoma on section and was filled with gelatinous, tough, cloudy-grey liquid, in which numerous actinomyces colonies were found on microscopic examination.

Buday's[125] patient, a female, aged thirty years, who had no cerebral symptoms, died as a result of chronic nephritis. At autopsy, a cystic tumour, about the size of a hazel-nut, suspended from the roof of the ventricle, was found between the pillars of the fornix and contained gelatinous tough material in which small actinomyces colonies were found on microscopic examination.

Sternberg's[126] patient, aged fifty-eight years, had suffered from headache for many years, and was diagnosed as suffering from a tumour of the third ventricle of the brain. At autopsy the lateral ventricles were found to be greatly distended, and in the third ventricle between the anterior and posterior commissures and attached to the anterior pillars of the fornix, a cyst, the size of a cherry, was found, which was connected with the lateral chorioid plexus. The cyst contained homogeneous, muco-gelatinous material, which was found on microscopic examination to contain numerous actinomyces colonies.

Hallervorden's[127] patient, aged sixty-four years, suffered from attacks of vertigo for seven months previous to her death. At autopsy the lateral ventricles were greatly distended, and a round tumour, about the size of a small walnut and resembling a myxoma on section, was found lying upon the *tela chorioidea* of the third ventricle. In the homogeneous basic substance of the mass various accumulations of cells in the process of disintegration and numerous colonies of actinomyces were found on microscopic examination.

Kroner[128] has recorded a case in which a gelatinous pear-shaped tumour containing actinomyces colonies was found between the laminæ of the *septum pellucidum*.

Melnikowa-Rasvedenkowa[129] has described a similar tumour in the third ventricle of her patient. Mucous colloid hyaline degeneration and actinomyces colonies were found on microscopic examination of the tumour.

In these six cases there is a marked similarity in the position and structure of each tumour and an apparent absence of another actinomycotic lesion in the body. The cyst membrane, except in one case in which it was definitely recognizable, is generally composed of poorly vascularized, partly cellular and partly fibrinous connective tissue. In all these six cases little or no inflammatory reaction was observed in the neighbouring brain substance, and no granules were recognizable on macroscopic examination of the gelatinous content of the cystic tumour. The "gelatinous necrosis" noted in each cyst may partly (and probably largely) be produced by toxins elaborated by the organism itself.

One interesting personal case, which will be reported later as a case of bilateral "excretory" actinomycosis, was seen in January, 1921. The apparent correct diagnosis of the patient's illness at that time was not made until November, 1929, when actinomyces granules were found in her urine. The patient, E.P., aged thirty-eight years, the mother of three healthy children,

sixteen, twelve and five years respectively, had suffered from scarlet fever at five years of age, measles at twenty, *la grippe* at twenty-three years of age, and an abscess in each breast, which developed after the birth of her second child. The abscess in the right breast healed rapidly after incision, but the abscess in her left breast, which was treated with "bluestone water", was very slow to heal. She had suffered from attacks of headache and vomiting for seven years, and during the previous eight days she had passed blood in her urine and also complained of pain in the right lumbar region. Early in the course of her illness headache, which was relieved by sedatives, occurred at intervals of about one month, and she vomited about one hour after the commencement of each attack. Gradually the intervals between attacks lessened, and for the previous fourteen days she was afraid to eat on account of vomiting. She had lost 6·3 kilograms (one stone) in weight during the previous six months. She had two previous attacks of hæmaturia; the first was slight in amount and lasted for two months while she was pregnant with her second child; the second attack occurred four months previous to examination, it was of three days' duration, and clots of blood were noticed particularly at the end of micturition. She did not experience pain or frequency in voiding urine, and occasionally wakened once or twice only at night during an attack of hæmaturia. Pain in the right lumbar region had been present for two days previous to the appearance of hæmaturia on the second occasion, and was quite absent, with the doubtful exception of one or two days on which she did the family washing, until she noticed blood in her urine eight days before her visit; during this time it had been constantly present and disturbed her sleep. She had worn glasses for two years without relief. On examination, it was apparent that she was a sick woman; she had a temperature of 37·8° C. (100° F.) and a pulse rate of 90. Her pupils reacted to light and accommodation, and she had paresis of the right external rectus muscle and slight papillœdema of the right optic disk; her knee jerks were slightly exaggerated, the other reflexes normal and no muscular atrophy or impairment of sensation was detected. The erythrocytes numbered 4,000,000, the leucocytes 7,800 per cubic millimetre. The hæmoglobin value was 80%. The polymorphonuclear cells numbered 72%, lymphocytes 24%, large mononuclear cells 3%, and eosinophile cells 1%. Examination of the urine showed the specific gravity to be 1,018; a trace of albumin was present; there was no sugar in the urine; there was an average of 120 erythrocytes in each microscopic field, and neither casts, crystals nor organisms were found. The cerebro-spinal fluid was under considerably increased pressure, and showed a slightly yellowish tinge, and contained four cells per cubic millimetre; the globulin test gave a "++" reaction and had a total protein content of 105 milligrammes per hundred cubic centimetres; the glucose content was 0·08%; no organisms were found on direct examination of smears or on cultivation of the fluid. Her blood and cerebro-spinal fluid yielded no reaction to the Wassermann test. An X ray examination of her head and a retrograde pyelogram revealed no abnormality. After three weeks' observation in hospital a diagnosis of cerebral tumour was made, the tumour not being definitely located. The hæmaturia disappeared three days after her admission to hospital, and although microscopic examinations were made at frequent intervals until May, 1924, blood cells were found only on one occasion in December, 1922. When she was leaving hospital, one of her relatives asked the resident medical officer to give her a prescription for medicine so that she would believe that something was being done for her condition. The prescription contained 0·6 gramme (ten grains) of potassium iodide in each half ounce of the mixture, and was ordered to be taken three times a day. Ten weeks after her discharge from hospital her cerebral symptoms gradually disappeared. Following a provocative dose of "Novarsenobillon", the examination of her blood and cerebro-spinal fluid was repeated, and again no reaction to the Wassermann test was obtained. She refused to have the blood of her children or husband examined, as she believed they were perfectly healthy. Potassium iodide was gradually increased until she was taking 2·4 grammes (forty grains) three times a day. From this time until May, 1924, she had only two slight attacks of headache,

which she attributed to worry. In June, 1925, she reported that she had suffered neither headache, backache or any other pain, and felt better than she had been for years, and intended to discontinue her treatment. Although hæmaturia recurred, as will be described in a later article, she has been free from all cerebral symptoms up to December, 1935.

Whatever the lesion in her brain has been or actually is now, the organism which caused the lesion and its toxins have been for fifteen years, and apparently still are, functionally latent, as occurs in certain cases of cerebral abscess (particularly those due to trauma), or possibly the lesion has spontaneously ruptured into the nasal or nasopharyngeal cavity without the patient's knowledge. This fact remains, that the symptoms of which she complained were relieved by treatment with potassium iodide, or probably, as occurs with actinomycosis in other regions, particularly those located in the cervico-facial region, the lesion has been "spontaneously cured" by the effects of time and the kindness of nature, possibly aided by potassium iodide. As a result of subsequent examinations I venture to suggest that her cerebral lesion was due to actinomycosis, which was proved definitely, almost nine years later, to be the cause of her "excretory" bilateral renal and bladder condition.

Although there is no definite proof that this patient actually suffered from cerebral actinomycosis, at the same time it must be admitted that an isolated lesion in the brain is conceivable in her case. The organism in some stage of its life history was possibly introduced after teeth extraction or other slight trauma. Having entered the body at some point in its external or internal surface, through a small lesion which healed, leaving no trace of infection or even without inducing pathological changes at its *portio invasionis*, it eventually entered lymphatic vessels and was carried by the blood stream to a region in the brain in which conditions were found favourable for colonization. The leucocytes, in which portions of mycelial filaments have frequently been observed, possibly acted as a vehicle for transporting the organism.

Jacoby,[189] in his review of the literature on cerebral actinomycosis up to 1925, found the upper portion of the digestive tract the point of invasion in seven cases of cerebral actinomycosis, and the respiratory tract in four; in ten cases he found it impossible to determine the point of invasion. The primary area of infection, that is, the site at which the actinomyces first induce pathological changes in the tissues (the point of invasion and primary area of infection may coincide), was found with certainty to be in the lungs in eleven cases, and in three cases this location was highly probable. Of the seven cases of primary cervico-facial location, four metastasized in round-about fashion by way of the lungs. Hæmatogenous dissemination usually occurred from the direction of the lung, and frequently from within the lung without any possibility of locating the point of entrance into the blood stream.

Jacoby further observed that often (seven cases) there was at the same time a direct extension of the actinomycotic process from the lung toward the outside, which did not reach the thoracic wall until after metastases of hæmatogenous origin had already become apparent. He determined the point of invasion into the blood stream with certainty in six cases (heart and large veins). In his collection the location of cerebral metastases was in the right side of the brain in seven cases and in the left in four cases. In five cases both hemispheres were attacked. In five other cases the case report merely stated "in the brain". All of the metastases were located in the pallium, and usually attacked cortex and medullary substance at the same time. The size of the metastases varied from the size of a lentil to that of a goose egg. In six cases the size was not definitely stated. In the remaining fifteen cases large areas (the size of a hazel-nut and larger) were more frequent. In five cases a rather large solitary abscess was present; in the remaining cases there were two, three or more abscesses.

Jacoby found that numerous usually small areas were not those in which a generous sowing of actinomyces was made possible by invasion into a large vessel or into the heart, but that this also occurred in those cases in which this form of invasion did not occur. In the majority of cases definite actinomycotic abscesses

were present. In one case there was an actinomycoma in addition to an abscess. In three other cases showing the formation of actinomycoma this neoformation was composed of a number of small degenerated areas. In two cases he found that metastatic small abscesses in the meninges ocurred simultaneously with the other abscesses, but in no case was there diffuse meningitis. He truly states that hæmatogenous metastatic meningitis is rare in actinomycosis. Actinomycotic meningitis generally results from the direct extension of cervico-facial, bucco-pharyngeal, sinus and middle ear infection and infection of the eyelids. Although infection from a focus in the upper nasal passages is possible, one searches in vain in available literature to find a definite case in which infection is reported from this hidden portal of entry. Rarely, it is an isolated lesion and is usually found, as one would expect, at the base of the brain and in the spinal meninges. Frequently mixed infection is present.

At least twenty-one definite cases of cerebral actinomycosis have been reported since Jacoby's article was published. In two reported by Barth[120] and Theissing[121] the temporal bone itself has been shown on microscopic examination of sections of the bone to contain actinomyces colonies. In the short report of a case reported by Jewell[122] the patient died within nine weeks of the onset of her illness, which commenced with sore throat and stiffness of the jaw. An extradural and also a larger subdural abscess developed in the right Rolandic area and were evacuated and drained. The pus was examined, and the presence of the "ray fungus" was revealed. No mention was made of a definite primary focus of infection.

Relatively few cases of actinomycotic infection of the spinal cord and its meninges have been recorded. The literature up to 1933 has been reviewed by Zaytseva;[123] the cases of Lesne and Belloir,[124] Cann and Hollis[125] and a case mentioned by Clough[126] have not been included. Zaytseva's patient developed a complete spastic paralysis of the lower extremities. The infection apparently resulted from direct extension of infection through the intervertebral foramina from a focus in the lung which apparently developed following an attack of pneumonia two years before his death. Although numerous series of sections of the spinal cord were examined, actinomyces colonies were found only in one preparation. In available literature only three other authors have described actinomyces colonies in the substance of the cord.

In a case reported by Stevenson and Dighton,[127] death was due to actinomycotic meningitis which followed chronic sphenoidal sinus infection, and occurred four days after excision of the right eye. Excision was performed at the patient's own request, and on account of panophthalmitis which followed an injury with a particle of burnt coke nineteen days previously. The patient had no symptoms of intranasal disease previous to operation.

In a *précis* of an article by Fabrio[128] mention is made of a case of meningitis in a congenitally syphilitic infant, aged nine months, which was due to an "anaerobic variety of streptothrix".

In the case of actinomycotic cerebro-spinal meningitis reported by Cann and Hollis the patient, aged nine years, died within fourteen weeks after the onset of symptoms which developed after the removal of adenoids. In two other reported cases the patient died after an illness of ten years' duration. The onset of symptoms in one case was associated with trauma. In some cases in which death has followed soon after the onset of symptoms indicating involvement of the cord or its meninges, an extensive area of granulation tissue with abundant development of fibrous tissue has been found in the vertebral canal, and often little or no infection of the meninges has actually taken place. A review of the literature clearly shows that the symptoms and signs of cerebral actinomycosis are not characteristic, and in many cases are similar to those produced by other pathological conditions within the brain or other associated conditions, as in Huday's case. When headache accompanied by defects of vision is complained of soon after the apparent onset of cerebral involvement, the patient is frequently sent to an ophthalmologist, as in the case reported by Ledeboer,[129] in which these disturbances were first believed to be due to the effects of chronic smoking.

In at least one case, that described by Becker,[140] a primary focus of infection was associated with an operation for empyema, and the sudden onset of severe headache was attributed to a blow which the patient had received on the head about the same time as headache occurred. The disease may be present without producing any signs or symptoms, and in other cases may pursue an acute, subacute or chronic course. To sum up the clinical features briefly, it may be stated that headache, moderate or severe, sometimes accompanied by vomiting and disturbances of vision, and occasionally a slight rise in temperature, is in most cases the only common symptom.

Diagnosis.

Most clinicians remember the possibility of the organism of actinomycosis being the cause of certain lesions in the head and neck region, but owing to the multitudinous variety and varied character of the lesions produced by the organism, and particularly when superadded infection is present, the real nature of the lesion is often overlooked, particularly when a foreign body is present or when the lesion has been preceded by external violence or when it has the appearance of a cyst or neoplasm. When the lesion presents the usual classical textbook clinical characteristics of "multiple sinuses discharging sulphur granules", the real cause of the lesion is rarely overlooked. Strange as it may seem, and although the region of the head and neck is clinically regarded as the most common site of infection, New and Figi have reported one hundred and seven patients with actinomycosis of the head and neck, examined at the Mayo Clinic, only seven of whom had been receiving treatment for actinomycosis at time of examination. In fairness, it must be admitted that some of the patients had not consulted a physician.

A few authors maintain that when iodide treatment has been commenced in suspected or unsuspected cases, or when secondary infection is present, microscopic examination of the lesions always fails to reveal the nature of the condition.

Other authors, despite iodide treatment "vigorously pursued", and even in the presence of secondary infection, have found typical actinomyces colonies on microscopic examination of sections of a tumour removed at operation or of purulent material obtained with or without surgical interference. However correct the statement of these few authors may be, the essential fact remains that the correct and definite diagnosis of the nature of the lesion, even if superadded infection is present and treatment has been commenced, is made only after colonies of the organism have been found on microscopic examination of suspected or unsuspected material. Repeated examinations by a competent bacteriologist may be necessary.

The presence of occasional or even numerous branched filamentous organisms is not sufficient evidence that the patient is or was definitely suffering from actinomycosis.

Serological tests—agglutination, complement fixation *et cetera*—and also dermal tests with culture filtrate occasionally give positive, but generally negative, results, and have rendered little or no service in the diagnosis of actinomycotic lesions in the region of the head and neck.

It must be remembered that the presence of macroscopic granules does not necessarily mean that the lesion is due to the organism of actinomycosis.

"Pearls" of degenerated malignant tissue, masses of fibrin, tuberculous *débris*, small foreign bodies *et cetera*, have been regarded as actinomyces granules until a microscopic examination has been made.

Eiken[141] has observed that other granules, having exactly the same macroscopic appearance as actinomyces granules, may occur in the same lesion. Microscopic examination and cultivation of the organism in these granules showed no evidence of the presence of the organism of actinomycosis which was found on microscopic examination and cultivation of other granules in the same lesion and which resembled these granules macroscopically.

When an abscess in the region of the head and neck is incised, it is essential to have an immediate systematic bacteriological examination made of the

maximum amount of purulent material that can be evacuated, and also, later, a careful histological examination of sections of various portions of any inflammatory or neoplastic mass removed at operation. A swabbing taken from a discharging sinus is almost invariably useless for diagnostic purposes.

Microscopic diagnosis is sometimes made more difficult by the fact that two or more diseases may coexist in the one lesion. A lesion produced by the organism of actinomycosis may precede, accompany or follow the development of a carcinomatous or sarcomatous lesion or even a tuberculous lesion in the same situation in a patient suffering from syphilis.

The clinical diagnosis is sometimes difficult when two or more diseases are present in different situations, and particularly difficult and sometimes impossible when near or distant metastases due to the organism of actinomycosis are also present.

In one case erysipelas developed in a lesion which was already the site of actinomycosis and carcinoma.

Prognosis.

The prognosis of apparently isolated cervico-facial actinomycosis is generally favourable. Most authors agree that there is a definite tendency to spontaneous recovery when the disease is situated in this location, particularly if the causal organism can be cultivated only under microaerophilic or anaerobic conditions.

The prognosis in isolated actinomycotic lesions in the maxillæ, mandible, tympanic cavity, pharynx or larynx is much more grave, and in the orbit and particularly in the brain almost hopeless. The prognosis is also almost hopeless in most cases in which the lesion in this region is a hæmatogenous metastasis from a recognized or unrecognized near or distant primary focus of infection.

Treatment.

The treatment of actinomycosis frequently tries the patience of both surgeon and patient, and sometimes the patient disappears without giving the surgeon a good opportunity to conduct and complete a special form of treatment. Even in accessible and limited areas of infection, surgical measures sometimes fail, and it behoves each one of us to report our apparent success or failure with surgical or surgical *plus* accessory treatment, in order that others in large cities and clinics, here and abroad, with greater facilities for thorough investigation, treatment *et cetera*, may try apparently successful methods of treatment, and either confirm or correct any observation made.

The internal administration of potassium iodide with or without surgical interference has been advocated for the treatment of this disease in man since 1892, seven years after it had been found to produce satisfactory results in the treatment of "wooden tongue" in cattle. An article published recently by McFadyean[142] suggests that "wooden tongue" in cattle in most, if not all, cases is caused by the actino-bacillus and not by *Actinomyces bovis*. Some cases have been reported in which iodides *per se* have caused definite improvement, and in other cases apparent cure for a number of years. Regardless of the excellent results claimed by a few writers, most authors believe that iodide cannot be regarded as an absolute specific treatment for actinomycosis, as it does not cure by direct action on the causal organism and must therefore act upon anatomical elements, tissues, glands *et cetera*, perhaps by increasing their resistance by an intimate mechanism which has so far escaped our observation.

Colebrook[143] cites the work of Jobling and Petersen, who express the view that "iodine neutralizes the action of the agents which prevent solution and absorption of necrotic tissue and at the same time lays bare to the action of the real germicidal agent the infecting organism which previously had been protected by the necrotic tissue". He suggests that favourable results from the administration of iodide in cases of actinomycosis are to be expected only when necrotic foci require to be broken down.

Many authors suggest that treatment should be commenced by the oral administration of two grammes of iodide daily, in water or in milk, the quantity

being increased rapidly or gradually to ten or even twelve grammes daily; they advise the continuance of treatment for some time after the lesion has been apparently cured. One author has suggested that it is necessary to use iodide of potassium in daily doses of at least ten grammes from the commencement of the treatment. Another author assures us that large doses of potassium iodide have a diuretic effect, which is an important factor in the prevention of iodism, and yet another writer believes that the presence of mixed infection retards or even wholly prevents the therapeutic effect of potassium iodide.

A number of authors advise sodium iodide to be given either orally or intravenously to patients with an idiosyncrasy to potassium salts.

Iodine and numerous proprietary preparations containing iodine, administered orally or intravenously, have been recommended by a few writers, sometimes in combination with other treatment.

Ionization treatment with 1% solution of potassium iodide has also been advised.

Darier and Gautier[140] advise electro-chemical treatment with 10% solution of potassium iodide. Two platinum needles, attached to the poles of a constant current battery, are inserted into the tumour, and fifty milliampères of current are passed through it, and every minute a few drops of the iodide of potassium solution are injected into the mass. The electric current decomposes the iodide solution into nascent iodine and potassium. A general anæsthetic should be administered, and the treatment repeated for twenty minutes every eight days.

Osborne[145] has shown that the normal iodine content of the cerebro-spinal fluid is increased following the oral administration of iodide, and particularly when iodide is administered intravenously.

Lesbouyries[146] states that iodine medication has little effect in actinomycotic lesions in bone.

Gradually increasing doses of 5% solution of "Yatren", commencing with five cubic centimetres and increasing to one hundred cubic centimetres, have been administered intravenously and frequently on successive days, and with apparently favourable result in an extensive inoperable lesion in the larynx after iodine and X ray therapy had apparently failed.

On account of the possibility of dissemination of the disease by a scalpel, electro-coagulation treatment combined with other measures has been recommended by a few authors.

Copper salts, administered internally and also applied locally in association with other treatment, have been advocated by a few writers.

Injection of a 1% solution of copper sulphate and a 20% solution of nitrate of silver into the lesions has also been suggested.

Intravenous administration of "Neosalvarsan" and also of 0·5% solution of gentian violet has helped to produce apparently good results in certain cases.

Methylene blue administered internally or injected locally in 3% solution, combined with X ray therapy, has also been claimed to have produced satisfactory results.

Local applications of iodine, ichthyol, carbolic acid, hydrogen peroxide and formalin in varying solutions have also been used.

It may be mentioned here that the only cases of actinomycosis involving the head and neck in this district in which death has occurred, have been due to an organism which was successfully cultivated only under aerobic conditions. One of these cases is included in this article. The causal organism in this case was not isolated until eight months after the patient first came under observation. Up to five years ago local treatment in cases of actinomycosis of the head and neck, when necessary, consisted of irrigation of the affected area with a 10% solution of hydrogen peroxide and exposure of the lesion to sunlight for increasing periods each day. Potassium iodide or tincture of iodine (French iodex) was administered orally in all cases.

Since that time solutions of formal-saline (formalin 40%, 10 cubic centimetres, and 0·9% saline solution, 90 cubic centimetres) have been used as a local application in cases in which the lesion was not completely excised; and

"Hexsotate" or hexamine in doses of 0·6 to 1·2 grammes (ten to twenty grains) has been administered orally three times a day and a careful examination of the urine has been made at weekly intervals.

When the diagnosis has been definitely established, no further attempt has been made to obtain material for cultivation on account of the danger of stimulating infection in any case in which the causal organism can be cultivated only under aerobic conditions.

In one extensive lesion involving the cervico-facial region, in which the patient refused X ray therapy, the lesion was excised in three stages, and two months later skin grafts were used to hasten the healing of the extensive granulating area left after excision. In a later article it will be shown that the surgeon has to be careful to prevent spread of infection with a scalpel, and, if excision of the lesion is performed in two or more stages, the application of formal-saline pads after operation appears to produce satisfactory results.

Colebrook[146] emphasizes the necessity of maintaining efficient drainage of abscesses which develop during the course of the disease, and recommends treatment with appropriate vaccines. He draws attention to the necessity for curetting infected tissues with a dry gauze swab, since by this means infected tissues will not be opened up, as is bound to occur if a sharp instrument is used. In his opinion infection will be promptly checked in every area that is efficiently drained, as the organism, although it can propagate in the fluid of an abscess cavity (which has lost much of its antitryptic power), cannot do so in unaltered lymph; he suggests that the hypertonic saline solution treatment advocated by Wright[147] should be adopted in order to promote a free flow of lymph through, and out of, the infected tissues.

Dean[148] has treated an actinomycotic lesion in the parotid region with four injections of vaccine containing twenty-five million mycelial fragments, at intervals of seven days, combined with incision, gauze curettage and iodides, as recommended by Colebrook. It may be mentioned here that Colebrook administered vaccine in doses of three to ten million fragments about once each week.

Neuber[34] [149] has contributed two interesting articles which show the value of specific vaccine therapy in certain cases of advanced abdominal actinomycosis. Weak and severely emaciated patients must be improved in general health and show specific allergic reactions before commencing treatment.

Intracutanenous inoculations with specific autogenous or polyvalent vaccine of high dilution resulted in specific focal, local and general reactions, while control injections of physiological solution of sodium chloride produced no reactions. Specific vaccine intracutaneous inoculation of control subjects also produced no reaction.

At the outset of treatment, in order to promote absorption of infiltrated tissue, the author employs gold therapy ("Solganal" preparations), at first intravenously and later intramuscularly at intervals of four to five days in increasing doses of from 0·01 to 0·5 gramme. Specific vaccine is then injected at intervals of four to five days in increasing doses of from 0·1 to 0·9 cubic centimetre.

Ten to fifteen injections of each are usually given, and, if they are not sufficient, the gold and vaccine treatments are repeated. Malarial therapy is sometimes combined with gold therapy in obstinate cases. He naturally prefers autogenous vaccine, but has used a polyvalent vaccine prepared from a number of different anaerobic strains when unable to cultivate the causal organism from the lesion. The vaccine is prepared from maltose agar cultures three to four weeks old. The colonies are washed off with five cubic centimetres of sterile solution of sodium chloride. This suspension is subjected to trituration for about fifteen minutes and is filtered; 0·5% phenol is added, and the solution is then stored in an ice-chest.

Treatment with actinomycine, a product obtained after filtration of broth cultures, has been employed in the treatment of cervico-facial cases in the Argentine Republic.[150] [151] Treatment is commenced with a weak solution of 1 in 100,000 concentration injected subcutaneously in quantities of 0·5 cubic centimetre. If the first injection produces no local, focal or general reaction, the

dose may be increased rapidly, first in quantity and then in concentration, until undiluted actinomycine is administered.

Epstein and Schoenholz[152] and a few others have, with apparently successful results, employed non-specific protein therapy in the form of typhoid-paratyphoid vaccine after treatment by intravenous injection of iodides.

Many authors are very enthusiastic regarding X ray therapy, and rightly point out the disfiguring scars which result from surgical treatment and also the grave danger of dissemination of the infection as a result of surgical interference, particularly when electro-coagulation methods have not been practised.

In Steinke's[153] case the cure of a facial lesion was attributed to a single X ray exposure to secure a Röntgenogram.

Stewart-Harrison[154] has reported the results of X ray therapy in twenty-two cases of actinomycosis in the region of the head and neck. When possible, potassium iodide was also administered. The treatment was apparently invariably successful. He contrasts the results of treatment by the disjuncted fractional method and the protracted fractional method; the latter method was found to be advantageous. It is characterized by the daily repetition of a small dose (30% U.S.D., 180 *r*/1) until a dose of 3,000 to 4,000 *r*/1 has been administered per field, whereby the duration of the application of each single dose is extended (by increase of the filter to 1·3 millimetres of copper and of the focus-skin distance to 60 to 70 centimetres) from a few minutes to approximately an hour.

Smith[155] collected from the literature one hundred and twenty-nine cases of cervico-facial actinomycosis, in most of which medical treatment in addition to X ray therapy had been given. The lesion was apparently cured in 83% of cases. He reports the treatment of seven cases of actinomycosis in the cervico-facial region, and recommends as an optimum dose 800 *r* of deep therapy (I.E.D.) generated at 200 kilovolts peak, and a filter of 0·5 millimetre of copper *plus* 4·0 millimetres of celluloid or the equivalent in some other filter. This dose should be administered at intervals of one to two weeks with the use of as many fields as are necessary.

Fantus[156] suggests that X ray therapy may be used for relatively superficial lesions in a dosage of 140 kilovolts, with 0·25 millimetre of copper and 1·0 millimetre of aluminium filtration. Such treatment may be repeated every two weeks for at least six months.

Lattes and Stuhl[157] advise small doses of X ray therapy at short intervals, and suggest that massive doses with high voltage at long intervals may have been the cause of failure in a case reported by Ravaut and Filliol,[158] in which the patient was later apparently cured after electro-coagulation treatment.

Palvarini[159] has reported a case in which he believes that excessive intense X ray therapy was the cause of an epithelioma which was associated with an actinomycotic lesion in the right zygomatic region.

Zwerg[160] points out the necessity for keeping patients who are undergoing X ray therapy under close observation, and also advises us to avoid curettage of the lesion; he emphasizes the danger resulting from the application of iodine to the skin of an irradiated area, and also mentions the detrimental effect upon the cosmetic result which arises from the application of iodoform after incision of an abscess. He administers higher doses than those recommended by Jungling, who suggested a minimum of 50% of a skin erythema dose. Zwerg distributes his doses over three to five sittings through heavy filters of 0·5 millimetre of copper and 1·0 millimetre of aluminium with a tension of 180 kilovolts and a current of four milliampères. A period of four to six weeks is allowed to elapse before the next series of treatments is commenced. In later stages of the treatment the doses are smaller, approximately 30% to 40% of the skin erythema dose.

Stuhl[161] points out that during treatment a violent reaction may occur which may cause treatment to be discontinued too early. He refers to the experience of Gougerot,[162] in whose case a violent reaction occurred on the day following the first treatment with X ray therapy and lasted for four days; a similar reaction occurred and lasted for two days after a second similar application. Two subsequent treatments produced no reaction. Gougerot believes that these

reactions are related to a phenomenon of biotropism which, far from contra-indicating the treatment, should cause it to be continued. In another article Gougerot, Dechaume and Giraudeau[165] describe in detail the X ray therapy in thirteen lesions involving the head and neck. They feel certain that treatment with radium will be found as effective as X ray therapy if sufficient quantities of radium are available.

Heyerdahl[164] reported the clinical history and results of treatment in twenty-one cases of infection of the face and neck. As soon as radium therapy was commenced, all other therapy was discontinued. In most of the cases radium γ rays from concentrated radium preparations were used and filtered through one to two millimetres of lead. Twenty-eight additional cases treated with radium at the same hospital were reported later by Engelstad.[165] The usual dose was 33 to 35 milligrammes of radium element, preferably with a filter of two millimetres of lead, applied for forty-eight hours. The number of treatments varied greatly. Preliminary incisions appeared to delay recovery.

New and Figi[111] published an extensive study of one hundred and seven cases of actinomycosis of the head and neck in 1923. In their experience, treatment with radium every three or four weeks, administration of potassium iodide in large doses, thorough drainage of purulent collections and packing with iodoform gauze so that the entire area was exposed to the air, caused the actinomycotic processes to disappear in all but very advanced cases in which there was extension to the meninges or thorax.

In two cases of actinomycosis, recently reported in New South Wales,[166] treatment was carried out with local applications of radium and the internal administration of potassium iodide. Our own experience of treatment with radium is limited to one case, in which the lesion was extensive and was similar to that recently reported by Gyarmati.[167] In Gyarmati's case a hard infiltrated mass, about the size of a fist, situated under the mandible, was reduced to a comparatively easily movable mass about the size of a green walnut, and this mass was excised without difficulty. The duration of the application was three days, and the dose of radium was 2,880 milligramme-hours. A two millimetre screen of silver was employed, and the radium was applied at a distance of two centimetres.

When we carefully examine the irregular results sometimes obtained from one or other of the many suggested forms of therapy or combinations of treatment advocated for the treatment of apparently "isolated" infection in certain areas in the region of the head and neck, we are amazed at the results obtained by other workers and occasionally by the individual advocate himself. This leads us to conclude that these varying results may be due to the fact that the suggested treatment has been only temporarily beneficial or that a different ætiological factor is present in certain cases; or they may probably be due to the presence of superadded infection or the metastasization of the infection in near or distant regions; or they may possibly be due to the coexistence of two or more diseases in the same patient which may or may not have been diagnosed during the life of the patient or even after death.

In interpreting the result after the employment of any or a combination of methods of treatment, it must be remembered that actinomycosis in the region of the head and neck occasionally undergoes complete subsidence.

We must remember, also, that it is absolutely essential to examine the patient repeatedly to determine whether the particular treatment adopted has been completely or partially successful, and also to determine whether an apparently cured lesion is recurring or extending to near or distant regions by continuity, contiguity, ingestion, inhalation, or by ductogenous, lymphogenous or hæmatogenous invasion.

The accompanying table comprises the cases observed in this district.

Appendix.

Medico-Legal Aspects.

In the available literature I am able to find only eight cases in which a medico-legal decision on the traumatic genesis of actinomycosis has been recorded.

TWENTY-SEVEN CASES OF ACTINOMYCOSIS INVOLVING HEAD AND NECK REGION.

Case Number.	Sex.	Age in Years.	Location.	Onset.	Duration of Symptoms.	Remarks.
1	M.	29	Right submaxillary region.	Followed extraction of a carious tooth.	11 months.	Incisions made in indurated area.
2	M.	32	Left naso-labial region.	Developed after treatment of a carious canine tooth.	4 months.	Two sinuses present in a localized violet-red indurated area which was excised.
3	F.	61	Left occipital region.	Alleged to be due to the frequent application of "Venice turpentine" to an abscess in this region.	17 months.	Lesion resembled a carbuncle with numerous discharging sinuses. Incised.
4	M.	4	Left submaxillary region.	Developed after a blow delivered accidentally by a playmate.	11 months.	Three sinuses in an infiltrated tumour discharged granules on application of pressure. Infected area excised.
5	M.	17	Right parotid region.	Associated with treatment of decayed teeth.	6 months.	Lesion resembled cellulitis. Incisions made in infected area.
6	F.	27	Left submaxillary region.	Followed extraction of several decayed teeth.	11 months.	Incisions made in tumour.
7	M.	36	Right cheek.	Alleged to have been caused by a bee-sting.	3 months.	Localized tumour about size of marble. Histological examination revealed presence of actinomyces colonies.
8	F.	29	Right upper eyelid and orbital margin.	Attributed to injury by a tennis ball.	9 months.	Infected area excised.
9	F.	34	Left submaxillary region.	Followed extraction of carious teeth.	11 months.	Tumour containing three sinuses excised.
10	M.	42	Left submaxillary region.	Attributed to carious teeth.	2 years.	Deep X ray therapy applied, followed by excision of tumour. Patient had epithelioma of lip removed five years previously.
11	M.	17	Lower left lachrymal duct.	"Sticky" substance exuding from left eye.	4 months.	Granules removed and 20% Argyrol drops instilled.
12	F.	29	Left submaxillary region.	Attributed to gumboils which had developed in her left lower jaw.	8 months.	Incisions made in tumour.
13	M.	32	Left parotid region.	Associated with extraction of two carious upper molar teeth.	4 months.	Lesion resembled a large abscess; granules found on incising abscess.
14	M.	33	Right cheek.	Followed treatment of a "pimple" in this region.	4 months.	Localized abscess excised.

TWENTY-SEVEN CASES OF ACTINOMYCOSIS INVOLVING HEAD AND NECK REGION.—*Continued.*

Case Number.	Sex.	Age in Years.	Location.	Onset.	Duration of Symptoms.	Remarks.
15	F.	36	Thyreoid gland.	Acute symptoms developed ten days after treatment of a goitre by a herbalist.	? years.	Microscopic examination revealed granules in cystic tumour to be actinomyces granules.
16	M.	46	Left lateral margin of tongue.	Followed injury with a cake fork.	15 weeks.	Microscopic examination showed presence of actinomyces colonies.
17	F.	29	Left submaxillary region.	Developed after extraction of carious teeth.	7 months.	Incisions made in tumour.
18	M.	26	Lower left central and lateral incisor teeth.	Alleged to be due to use of new toothbrush.	3 weeks.	Three teeth extracted and cavities irrigated twice daily with peroxide of hydrogen, followed by tincture of iodine.
19	M.	56	Left margin of lower lip.	Tumour following injury to lip with a tobacco-pipe.	9 weeks.	Microscopic examination revealed actinomyces colonies in tumour, which was diagnosed clinically as epithelioma.
20	M.	52	Floor of mouth, lip and cervico-facial regions.	Alleged to follow treatment of infected cow.	2 years.	Multiple incisions made, which appeared to stimulate infection; later proved to be due to an aerobic actinomyces. Patient died.
21	M.	27	Lower left cervical region.	Wound failed to heal following excision of "tubercular" gland.	10 months.	Incisions made in tumour and application of formal-saline to gauze inserted into wound.
22	F.	32	Right submaxillary and cervico-facial regions.	Followed extraction of carious teeth.	7 months.	Large tumour removed in three stages, each operation area covered with gauze, which was moistened daily with formal-saline.
23	F.	34	Lower left lachrymal duct.	Inner canthus of right eye became swollen, accompanied by a slight discharge.	13 weeks.	Granules removed, and Argyrol drops instilled.
24	M.	52	Left submaxillary and cervical regions.	Followed treatment for pyorrhœa and filling of decayed teeth.	11 months.	Radium applied to an indurated tumour, the size of a hen's egg. Later the remaining portion of tumour excised.
25	F.	51	Lower right lachrymal duct.	Developed after removal of foreign body on conjunctiva.	6 weeks.	Granule removed and Argyrol drops instilled twice daily.
26	M.	26	Right cervico-facial region.	Attributed to discharge from the orifice of a branchial fistula.	14 months.	Sinuses incised and formal-saline pads applied. No communication with branchial fistula observed.
27	M.	23	Right parotid region.	Followed dental treatment of infected teeth.	7 months.	Four incisions made in tumour and packed with formal-saline gauze, which was moistened every three hours.

Noesske[105] and, later, Küttner[169] have reviewed the literature on traumatic actinomycosis up to 1922 and 1932, respectively. The cases recorded by Douglas-Crawford,[170] Downes,[33] Bartoli,[112] Reckzeh,[171] and Allodi[172] have not been included.

In view of the fact that compensation acts are in force in most civilized countries of the world, the association of actinomycotic lesions with trauma becomes of paramount importance from a medico-legal aspect. In the past, too little attention has been paid to the possible association of trauma with infection by the organism of actinomycosis.

Küttner states that there are three possibilities to be considered: (i) direct implantation of the organism into the wound, (ii) secondary migration of the organism to the injured area, (iii) latent infection becoming manifest as the result of trauma.

In a case reported by Thiem, witnesses were able to verify the fact that a farmer, while unloading straw, accidentally aspired a fragment into his throat. He endeavoured to expel the fragment by coughing and vomiting, and finally apparently managed to swallow it with the aid of a piece of bread. The lesion was diagnosed as actinomycosis four months later. The Co-operative Vocational Association admitted that the lesion resulted from injury.

A number of cases, not the subject of litigation, could be mentioned, in which a foreign body (portions of Gramineæ, splinters *et cetera*) either acted as a direct carrier of infection from without or by its traumatic and irritative effect caused infection by the organism of actinomycosis already present in the oro-pharyngeal cavity of the patient.

In Loxton's[31] case, a bricklayer fell on a kerbstone and sustained a scalp wound in the left parietal region, which was subsequently followed by the development of an actinomycotic lesion. Noesske regarded the kerbstone as the source of infection, because such stones may be soiled with street sweepings containing straw and chaff.

In another case actinomycosis developed in the parotid region at a point at which a man had been injured with a manure fork; Noesske believed that plant particles infected with the organism of actinomycosis must have been present on the manure fork and transmitted the infection to the patient. It should be mentioned that two mules suffering from fistulæ of the jaw may have been the primary source of the infection.

In the second type of traumatic actinomycosis, gross injury is usually associated with fracture of the maxilla or mandible. In one case recorded by Küttner the patient, during the progress of a hunt, struck himself on the chin while leaping over a ditch. The gun went off and the entire loading of shot penetrated the left side of his face, causing severe laceration of the soft tissues and multiple fractures of the maxilla and mandible.

In another case, a boy, aged eleven years, developed actinomycosis following a compound fracture of the jaw.

With reference to compensation, Küttner states that such cases of secondary actinomycosis which are usually more severe than the primary injury should be recognized as the results of the injury.

Compensation was granted in one case in which a farmer's wife had been kicked in the mandibular region by a cow. The actinomycotic nature of the lesion was recognized three and a half months after the injury.

I am unable to find a definite example of the third type of traumatic actinomycosis in any of the reported cases involving the region of the head and neck.

In two cases in which the left foot and lower part of the left leg respectively were the seat of a traumatic actinomycotic lesion, Küttner states that, if called upon to pass a verdict, he would recognize the connexion between the lesion at the second time of observation and the primary trauma which had occurred in the one case thirty-seven and in the other case seventeen years previously.

The provisions of the *Workers' Compensation Act* of New South Wales define injury as personal injury arising out of and in the course of employment, and includes any disease so arising, whether of sudden onset or of such a nature as

to be contracted by gradual process. It will, therefore, be observed that the applicant for compensation must prove that the injury arose out of and in the course of his employment; this is a very difficult matter to prove, particularly in cases of actinomycosis of the head and neck, and especially when the lesion is in or near the oro-pharyngeal cavity. The facts may be that the patient's disease is definitely diagnosed as actinomycosis, but whether the applicant can prove his claim to the satisfaction of a court of law depends upon the nature of the facts that he can adduce to establish the causal relationship between a set of circumstances under which medical opinion would be certain beyond doubt that the patient had become infected with the organism of actinomycosis at his employment and the actual presence of the disease at a later stage in the worker's history.

Summary.

Twenty-seven cases of actinomycosis, involving the head and neck region, that have been observed in this district during the past twenty years have been tabulated. Available literature up to the end of 1934 has been reviewed.

"Actinomyces" is not a type designation, but a genus concept which includes a large number of types and varieties with highly varied morphological and biological characteristics.

There are both pathogenic and non-pathogenic aerobic, microaerophilic and anaerobic actinomyces.

The morphology varies considerably even in the same type.

The clinical picture of actinomycosis involving the head and neck and also autopsy revelations are not characteristic.

More cases will be diagnosed correctly if the surgeon or clinician is "actinomycosis conscious".

The medico-legal aspect of the disease is referred to in the appendix.

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Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

THE USE OF THE "DONATI INSTRUMENT" FOR THE CLOSURE OF DUODENAL OR SMALL INTESTINAL STUMPS.

Dr. Fritz Strauss: *Zentralblatt für Chirurgie*, December 7, 1935, Number 49, page 2910.

THE crushing clamp which is known as Donati's instrument, has, in contrast to the suture machine of V. Petz, the undoubted advantage of handiness. A further advantage is the smallness of the apparatus, which can be used with little manipulation in the resection of the stomach. The instrument illustrated in Figure I consists of two comb-like blunt blades, of which the lower one in the picture is constructed as a travelling blade. On each blade is a groove, which allows the passage of a needle.

When the Donati clamp is used, a ribbon intestinal clamp is applied to the duodenum close to the Donati clamp, and the division is made between the clamp and the instrument (Figure III). The instrument is fixed by pressing down a spring in the handle. The end of the bowel, now partially crushed and pleated

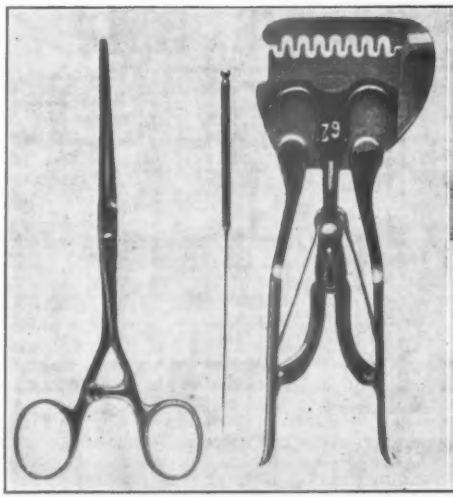


FIGURE I. The essential parts of the Donati instrument.

FIGURE II. The application of the open instrument to the intestine.

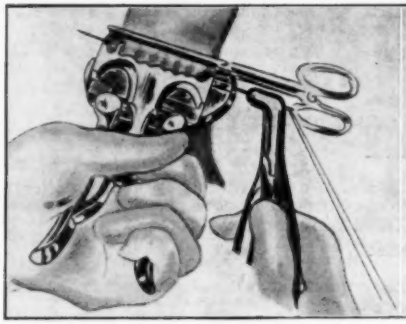


FIGURE III. The instrument is applied to the intestine and closed; a straight needle on a needle-holder is pushed through the hole running through the blades of the instrument, the pleated and cut end of the duodenum thus being transfixed.

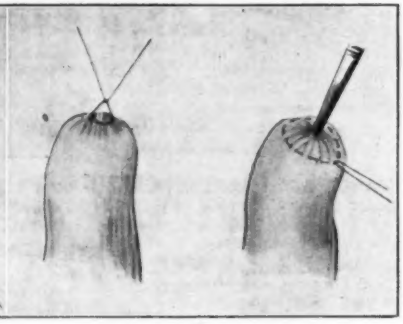


FIGURE IV. The instrument is removed, the thread which was attached to the needle is tied, and the intestinal end is embedded with a purse-string suture.

by the instrument, is transfixed by a needle armed with a suture which is pushed through the hole provided in the instrument. When the instrument is removed and the thread is tied, the suture of the stump follows in the usual way.

THE TREATMENT OF CANCER OF THE TONGUE.

L. Zukschwerdt and O. Fuchs (Heidelberg): "*Zur Behandlung des Zungenkrebses*", *Der Chirurg*, October 15, 1935.

AT Heidelberg, in the Samariterhaus, carcinoma of the tongue is treated by radium or deep X ray therapy, or by both combined with operation. In Professor Enderlen's clinic, with which the Samariterhaus is closely associated, all patients with carcinoma of the tongue are operated on. An investigation of the cases of this nature treated in these two clinics is of particular interest, because it enables a clear comparison to be made between the radiation and the surgical treatment of carcinoma of the tongue.

Zukschwerdt and Fuchs have collected the results over a five-year period of the treatment of cancer of the tongue in these two departments. The cases comprise 22 which were treated by operation, 15 in which radium or X ray treatment was employed in conjunction with operation, 59 in which X ray or radium treatment or both were used, and 18 cases of recurrence of carcinoma of the tongue which were treated with radium or deep X ray therapy.

The operation for carcinoma of the tongue as carried out by Enderlen was ligature of the external carotid artery and removal of the glands of the neck and half of the tongue in one sitting. In two cases the whole of the tongue was removed, but these cases, the authors state, should have been regarded as inoperable. Palpable glands were present in about 70% of cases. In 25% of these, when the enlarged glands were removed and subjected to histological examination, no cancer cells could be demonstrated. This fact, the authors hold, must be regarded as important in forming a judgement on the results of the radium treatment of glands. They quote Simmons as stating that in 56% of suspected carcinomatous glands he found no cancer; but that, on the other hand, in 20% of cases in which the glands were not palpable he found carcinoma.

Only in the presence of palpable glands have the authors made the block dissection of the neck, and then only on one side.

In the statistics they have tried to find out and to compare the results of radiation treatment only (radium and X rays), of surgical treatment only, and of surgical treatment in combination with the radiation treatment.

The immediate mortality from operation treatment was comparatively high —18·2%. This, they explain, was due to the fact that probably many of the cases should have been regarded as inoperable. They point out that pure radium treatment was not free from immediate mortality. In their series 10·2% of patients treated with radium alone died after the treatment from bleeding, pneumonia and other causes.

As shown in the tables, the number of five-year cures following operation was 31%. Two of these cases they regarded as inoperable, but they included them, so that there could be no doubt whatever that they had taken what they call the "absolute" figures. They criticize the radiation statistics of the treatment of cancer of the tongue as given in literature. They point out that many of the patients treated by radium are regarded as cured when the original tumour of the tongue has disappeared; that most of these statistics are based on a three-year cure, and many of the patients died between the third and the fifth year; that there are very few five-year cures unless radiation treatment has been combined with surgical treatment, and it is then difficult to say how far radiation treatment and how far operation has been responsible for the result. They state that the recurrence after operation can always be held checked by radium treatment, but that radiation can make no impression on the recurrence after radiation treatment.

They come to the conclusion that surgical treatment gives far better results than pure radium treatment, and that irradiation after surgical treatment has not increased the number of cures. They have no doubt, however, that it prolongs the life of the incurable patient. They find that no proof has been brought forward that the radium treatment can be in any way regarded as the method of choice in the treatment of carcinoma of the tongue; and that radium treatment without surgery is not to be considered.

The results described by these authors are set out in the accompanying tables.

TABLE I.

Pure Surgical Treatment.

Operation.	Number of Cases.	Post-Operative Deaths.	Deaths from Recurrence or Metastasis.	Deaths from Some Other Cause.	Symptom-free after Five Years.
Total extirpation, with dissection of the glands	2	ddkk 2	0	0	0
Removal of half of tongue, and dissection of glands	11	mndk 2	dik 5	dg 1	cd 3
Excision through sound tissue, and dissection of glands	o 5	 0	l 1	 0	ae 3
Excision through sound tissue without dissection of glands ..	4	0	1	fh 2	b 1
Total number of cases	22	4 (18·2%)	8	3	7 (31·8%)

a = Resection of the middle piece of the lower jaw. b = Dead of heart failure after ten and a half years (*post mortem* examination showing no metastasis). c = Dead of heart failure after seven and a half years (*post mortem* examination showing no metastasis). d = Transverse splitting of the cheek. e = After nine years dead of arteriosclerosis (no autopsy). f = After three years, dead of apoplexy (*post mortem* examination showing no metastasis). g = After one year, sudden death, accident, had no *post mortem*. h = After two and a half years, sudden death, accident, no metastasis. i = Operation on recurrence of primary tumour removed five years previously. k = Temporary jaw resection. l = After half a year, removal of a local recurrence. m = Recurrence of tongue gangrene, general infection. n = Pneumonia.

TABLE II.

Operation and Prophylactic After-Treatment.

Type of Treatment.	Number of Patients.	Died During Treatment.	Symptom-free After Five Years.
Excision + X rays + radium	8	1	1
Excision + X rays	3	0	2
Excision + radium	4	1	1
Total cases	15	2 (13·3%)	4 (26·6%)

TABLE III.

Pure Radium Treatment.

Type of Treatment.	Number of Patients.	Died During Treatment.	Symptom-free After Five Years.
X rays	15	2	2
Radium	5	1	0
X ray + radium	35	3	2
X ray + thorium	2	0	0
X ray + radium + thorium	2	0	0
Total cases	59	6 (10·2%)	4 (6·8%)

TABLE IV.

Pure Operative Treatment with Post-Operative Recurrence.

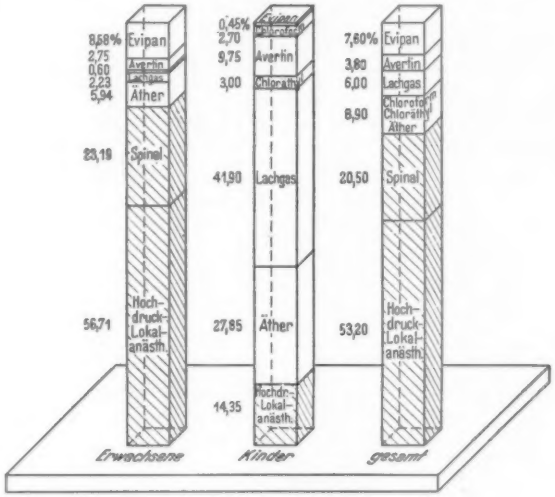
Type of Treatment.	Number of Patients.	Died During Treatment.	Symptom-free After Five Years.
X rays	5	0	0
Radium	5	1	0
X rays + radium	7	2	0
X rays + thorium	1	1	0
Total cases	18	4 (22·2%)	0 (0%)

ANÆSTHESIA IN KIRSCHNER'S CLINIC AT HEIDELBERG.

Dr. D. Philippides: *"Die Art der Schmerzausschaltung bei Operationen in der chirurgischen Universitätsklinik Heidelberg"*, *Der Chirurg*, January, 1936, page 13.

THE accompanying tabulation, showing the types of anæsthesia used in Professor Kirschner's clinic at Heidelberg, must strike Australasian surgeons as unusual. The left hand column refers to operations on adults, the middle column to children,

and the right-hand column shows the various methods for patients of all ages. One must immediately be struck by the limited use of ether for adults, and the very much greater use of nitrous oxide (*Lachgas*) and "Avertin" for children as compared with their use for adults. In the text of the article the author



develops the theme that "the Kirschner clinic has for many years led the fight against the use of general anæsthesia"; he also states that local (including spinal) anæsthesia is now used in 80% of operations on adults. This general plan for anæsthesia is as follows:

Head	Brain	Local (1% "Novocain").
	Face	Local.
		Local with intravenous narcosis.
		Rectal anæsthesia with intravenous narcosis.
	Mouth	Local.
		Rectal "Avertin" + chloroform, ether, and intravenous narcosis.
Neck		Local.
Thorax	Chest wall	Local.
	Lung	Pressure ether or gas.
Abdomen	Upper	Exclusively spinal.
	Lower	Spinal, ether, gas, or local, with intravenous narcosis.
Hernias		Local.
Kidneys		Spinal.
Bladder		Spinal or local.
Prostate		Local (pre-sacral).
Extremities		Spinal, local, or gas.
Spine		Local.

Intravenous narcosis in this table indicates the use of scopolamine with "Eukodal" and "Ephetin". In *Der Chirurg* of July 1, 1935, page 451, Philippides writes on this subject at greater length. The solution is scopolamine 0·0005, "Eukodal" 0·01, and "Ephetin" 0·025 gramme per cubic centimetre; the solution is put up in sterile ampoules by Merck. One cubic centimetre is given slowly, at the rate of 0·5 cubic centimetre per minute. This method greatly extends the sphere of spinal and local anæsthetics, and it is very valuable when used before

gas or ether. In comparison with the usual morphine and atropine it has the advantage that its action is immediate and is more powerful than the hypodermic injection, while the scopolamine has an action similar to that of atropine in checking bronchial secretion.

Spinal anæsthesia is, of course, given by Kirschner's air-replacement method, which Philippides considers is ideal for all major abdominal operations.

For local anæsthesia the solution used is 0·5% "Novocain", with adrenalin, and amounts up to 300 cubic centimetres are injected without fear. Kirschner has devised a high-pressure injector, which is used entirely to replace the use of a hand syringe. The solution is forced into the tissues under a pressure of two to two and a half atmospheres, the needle being kept stationary during the injection. The fluid forces its way along the various connective tissue planes, and from a single puncture produces a complete block in the limbs. The site of the skin incision must be infiltrated as usual. In 11,000 such procedures only one fatality has occurred; this was due to a fault in technique, not to the procedure itself. The danger of the solution entering veins has been shown not to exist. All brain, skull, neck and thoracic wall operations, as well as all perineal prostatectomies, have been done by this method exclusively.

The use of "Evipan" is limited definitely to short procedures. It was used earlier for longer operations, such as mastectomy, fractional doses being given during the operation by means of a motor-driven apparatus devised by Kirschner; but this is now entirely given up as dangerous. Philippides thinks that "Evipan" is usually given too quickly. After a slow injection of from four to five cubic centimetres, a pause of one minute is made, and thereafter more solution is added at a rate of not more than two cubic centimetres per minute, until the desired depth of anæsthesia is reached. Toxic patients take "Evipan" badly, and it must be given to them with great caution. Two to three cubic centimetres are often sufficient to produce deep sleep in a toxic patient, and the after-drowsiness is pronounced. "Eunarcon" does not seem to have many advantages over "Evipan", and it has its own disadvantages.

"Avertin" is used as a basal narcotic only, anæsthesia being completed by some more easily controllable medium such as ether, gas, chloroform or "Evipan". Of 119 cases in which it was used, "Evipan" or "Eunarcon" was added in 53, chloroform in 40 (38 of the 40 patients were children), gas or ether in four, and it was used alone in 22 cases (of the 22 patients three were children). The dose is based on 0·1 to 0·125 gramme per kilogram of body weight for children, and 0·08 to 0·1 gramme per kilogram in adults; it is given in a 3% solution. Philippides thinks that "Avertin" used in this way is a satisfactory and safe basal narcotic for children.

STENOSING TENOSYNOVITIS.

R. Soeur (Bruxelles): *"La ténosynovite sténosante", Revue d'orthopédie et de chirurgie de l'appareil moteur*, Number 3, 1935, page 193.

IN 1895 de Quervain first described a thickening and stenosing of the extensor synovial sheath for the tendon of the long abductor and short extensor of the thumb in their osteo-fibrous canal over the styloid process of the radius (Figure I). Soeur describes eight cases and refers to the paucity of attention to this condition in France particularly, although Winterstein's monograph in German (*Münchener medizinische Wochenschrift*, 1927, Volume lxiv, page 351) and Finkelstein's in English (*Journal of Bone and Joint Surgery*, 1930, Volume xii, page 509) are important contributions.

The pathological lesion is an hypertrophy of the dorsal carpal annular ligament over the radial styloid, and it is this that strangulates the two tendons, causing limitation of movement and pain in the affected thumb. The rôle of injury seems certain. Of the eight patients reported, seven gave a history of injury. In one case the development of this condition was directly blamed to knocking the external border of the wrist on the lid of a casserole dish. On the other hand,

small repeated traumata (microtraumatism) which daily pass unperceived, such as occur in pianists, cabinet-makers and especially housewives between forty and sixty years of age, are common. The classical clinical features are as follows: A female, about fifty years of age, may have a visible swelling "*de la tabatière*"; flexion-opposition of the thumb causes pain over the radial styloid (first pathognomonic sign), palpation over the same area is very painful (second

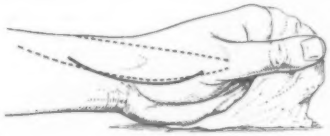


FIGURE I.

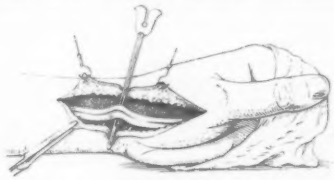


FIGURE II.

pathognomonic sign), and X ray examination always fails to reveal an abnormality. In the treatment, physiotherapy is uncertain; Hoffmann is the protagonist of immobilization of the thumb for several months. The procedure of choice is surgical intervention, because fifteen days after operation the patient can return to work. The technique is simple. Under "Novocain" infiltration of the snuff-box area and of the radial styloid, an incision five to six centimetres in length is made (Figure I) along the radial border of the thumb. The radial nerve is avoided. The aponeurosis and tendon sheath are slit for about 2·5 to 5·0 centimetres (one to two inches), then the skin is very carefully sutured, because there is no superficial fascia to suture and, of course, no sutures are inserted into the incised carpal ligament. No splinting is necessary. Movements are initiated as soon as possible (almost immediately). The results of the operation are very satisfactory.

Reviews.

OPERATIVE GYNÆCOLOGY.

Operative Gynæcology. By DR. H. V. PEHAM and DR. J. AMREICH. Volumes i and ii. London: John Bale, Sons and Danielsson. Sydney: Angus and Robertson. 1934. Pp. 779. Price: £7 10s.

THE late chief of the I. Frauenklinik of the University, Vienna, and his first assistant have produced in two magnificent volumes the finest work on modern gynæcology since Howard Kelly's classical textbook of thirty-five years ago. This production from the Peham Klinik is divided into three parts.

In the first place, there is a description of the methods and materials which are required in the preparation for, and in the successful performance of, any gynæcological operation.

Secondly, as one would expect from such a distinguished continental medical centre, there is a large and valuable section devoted to the surgical anatomy of the female pelvis, where the pelvic connective tissue, the topography of the ureters, the blood vessels and the nervous supply are described and illustrated in greater detail than in any other book of the kind. Much original work and many dissections were carried out by the authors, and these are reproduced in a very large number of beautiful pictures and coloured plates.

Thirdly, there follows the part on operative technique. Here the operations practised at this famous clinic are also described with a wealth of both important and practical detail, again accompanied by a truly wonderful profusion of life-like coloured drawings. The authors make a profound appeal to the surgical judgement of their readers when they discuss fully the indications for each operation and in particular when they relate their own experiences and final results.

Praise should be given to Dr. L. Kraeer Ferguson, Associate in Surgery at the University of Pennsylvania, for his laborious task of translating the text into English.

The opening chapter deals fully with the requirements of a good operating theatre, and all the necessary and careful preparations for an operation are described in detail.

The remarks on local anæsthesia are both instructive and interesting, because local anæsthesia in gynæcology is not commonly employed in English-speaking countries. The direct infiltration of the Schuchardt incision is given in detail, also for the excision of Bartholini's cyst. Block infiltration of the perineum is also clearly illustrated by anatomical pictures. Pre-operative and post-operative treatment are discussed in a full and practical manner.

In the Peham Klinik all operable cases of epithelioma of the cervix are treated surgically, and only inoperable cases are treated by radium and deep X ray therapy. The vaginal operation of the authors follows that of Schauta, but the extirpation of the uterus with the surrounding parametrium is much wider and more radical. A Schuchardt or vagino-perineo-levator incision is first made to gain full and complete access to the cervix. The subsequent radical vaginal hysterectomy can be followed in every detail by the clear description and by the many beautiful coloured illustrations. After the uterus is removed the peritoneum is carefully drawn together, because complete closure of the peritoneal cavity gives the best prophylaxis against post-operative peritonitis. Because of the definitely wider and more radical removal of parametrium, and in spite of having made a larger number of cases operable, the authors are gaining 20% of absolute cures. An interesting comment is made that prophylactic use of radium inserted vaginally after operation is improving even upon these results.

Then follows a description of the radical abdominal operation by Latzko's method, which is even more radical than Wertheim's. Every detail of this operation is again clearly described and richly illustrated by numerous coloured plates. With the removal of the uterus and surrounding tissues, the pelvis is now completely empty. So much of the pelvic peritoneum is excised that it is usually impossible to re-cover the wound cavity. The movable loop of the sigmoid is therefore used to close this peritoneal defect.

Wertheim's operation is also described and illustrated in full detail.

The authors' remarks on carcinoma of the cervical stump are both interesting and original.

Carcinoma of the *corpus uteri* is rightly treated by pan-hysterectomy, and, naturally, the authors' choice is by the vaginal route.

Simple vaginal hysterectomy is favoured for functional uterine bleeding (metropathia); this method, rather than the abdominal, is not followed out often enough in English-speaking countries.

The usual surgical treatment of myoma is also well described and illustrated, and particular attention is paid to the authors' method of treatment by vaginal pan-hysterectomy, in which the anterior lip of the cervix is slit up, the tumour or tumours being then removed by morcellation and finally the uterus.

Extrauterine pregnancy is treated fully, and the operative measures are, as usual, profusely illustrated. When the diagnosis is in doubt, an interesting and a true comment is made that a posterior colpotomy is preferable to, and gives more information than, needling the posterior fornix.

Conservatism is wisely advocated in treatment of pelvic inflammatory disease, and surgery is a final resort, when reasonable conservatism should again be displayed.

There is an interesting chapter upon genital tuberculosis, which is comparatively rare in Australia. The surgeons at the Peham Klinik exercise conservatism both in advising and in performing an operation.

In the surgical treatment of prolapsus, the choice of operation depends upon the patient's age. If the patient is in the fertile period, simple plastic operations are favoured, such as anterior colporrhaphy, with or without amputation of the cervix and perineorrhaphy. In older women, the interposition operation is per-

formed. In British and Australian clinics this operation has been abandoned, and for the various types of genital prolapse the Manchester or Fothergill operation, with modifications, has been adopted extensively.

Schubert's vaginal plastic operation for the formation of an artificial vagina is fully described and illustrated.

The surgical treatment of bladder fistulæ is given in detail and with much practical advice. The authors, however, feel that in ureteral fistulæ, if the lesion of the ureter is not completely transverse, a spontaneous closure may and does occur.

The authors have done much original research on the surgical treatment of urinary incontinence, and their surgical work for the relief of this commonly neglected condition is most valuable and convincing.

Kehrer's operation for vulvar carcinoma and the radical excision of deep and superficial inguinal glands are fully described and, as usual, beautifully illustrated.

In this book there is a curious absence of reference to the surgical treatment of chronic cervicitis by resection or amputation of the cervix; it is mentioned only in the surgical technique of genital prolapse.

Finally, this magnificent textbook cannot be too strongly recommended to every gynæcologist, who will be continually refreshed, rewarded and entertained by repeated study of the surgical anatomy and surgical technique which are so richly written into this work. This book is going to be just as necessary and invaluable to a second group of readers, younger and inexperienced, but nevertheless eager to perfect themselves from this essentially practical but truly scientific work.

THE FOOT.

The Foot. By NORMAN C. LAKE; 1936. London: Baillière, Tindall and Cox. Medium 8vo., pp. 330, with 94 illustrations. Price: 12s. 6d.

IN this the concrete age and the era of standardized footwear, when man is expected to fit his shoes, it is not surprising that foot complaints have increased in frequency, and the subject, therefore, in importance. A book such as Mr. Norman Lake's, dealing with the foot from the standpoint both of theory and practice, is very welcome. In its pages we find an account of the evolution, development, anatomy and physiology of the foot. This is followed by an interesting chapter on footwear, in which the effects of high heels are clearly explained. Then follow chapters in which the abnormalities, injuries and infections of the foot are adequately discussed.

It has been realized of recent years that certain minor abnormalities are important factors predisposing to functional failure of the foot, and Mr. Lake gives some interesting tracings depicting the variations of the curves made by the points of the toes; these variations he shows are of the utmost importance in determining the onset and localization of many of the effects of foot strain, as well as the production and localization of callosities and corns.

In the chapters on flat foot and *hallux valgus* the modern views about these conditions are excellently expounded, and the evidence for them is contrasted and compared with that for the older theories. In the latter, the longitudinal arch is viewed as a static structure dependent for its maintenance, when the foot is inactive, on the bones which are so conformed as to produce an architectural curve with the head of the astragalus as its keystone, the arch being supported by the soft structures and in the active foot by muscular action. Modern ideas do not hold with the existence of a structural arch in the natural foot which has never been restricted by footwear. They regard the living foot as being pulled up by the postural tonus of the muscles with the foot inactive, and this is replaced in the active foot by contractural tonus. The ligaments are regarded as of but little importance.

Hallux valgus is not purely a local deformity, but part of a more general derangement of the foot.

In further chapters, corns, affections of the nails and many miscellaneous conditions are attractively dealt with.

This book fills a real need, and can be strongly recommended; with none but very minor points do we disagree.

AN ATLAS OF ANATOMY.

Baillière's Synthetic Anatomy. By J. E. CHEESMAN, with foreword by J. E. Frazer, F.R.C.S.; 1936. London: Baillière, Tindall and Cox. Crown 4to. Price: 45s. net.

THE latest part of Baillière's well-known "Synthetic Anatomy" deals with the eye and orbit. The author has selected the various planes with care, and the twelve sections presented cover all the intricacies of this region in a lucid manner. The sections are taken in a sagittal plane from the lateral aspect towards the mid-line.

The whole of this series of sectional anatomy is now complete in fourteen parts bound in a loose-leaf cover. It will be particularly useful to the student who uses the method of building up his knowledge of anatomy from a basis of osteology and who draws in the superimposed structures.

To a busy surgeon who wishes to look up a special anatomical point, the atlas may be slightly confusing and may not give such a composite impression as he would desire. We can recommend the series as an adjunct to the dissecting room and blackboard.

MIDWIFERY IN THE TROPICS.

A Textbook of Midwifery in the Tropics. By V. B. GREEN-ARMYTAGE and P. C. DUTTA; Second Edition; 1936. London: Butterworth and Company (India), Limited. Australia and New Zealand: Butterworth and Company (Aust.), Limited, Sydney, Melbourne, Wellington and Auckland. Demy 8vo., pp. 425, with 40 illustrations. Price: 21s. (Aust.).

THE second enlarged edition of "A Textbook of Midwifery in the Tropics", by Green-Armytage and Dutta, contains much of interest to the Australian reader. Throughout the book emphasis has been laid more on clinical diagnosis and treatment than on theories.

In the description of ante-natal supervision stress is laid on the value of regular estimation of the ante-natal weight of the mother. The chapter on the toxæmias of pregnancy is well written, and conservative lines of treatment are advocated. Obviously obstetrics presents a serious problem in India, as elsewhere. Pyelitis is very common, and the authors consider that its prevalence may be due to the almost constant presence of intestinal parasites among their patients. A chapter on the anæmias of pregnancy and osteomalacia is most instructive, especially because of their relationship with vitamin deficiency. The authors advocate the routine administration of quinine during the last six weeks of pregnancy because of the lessened chance of fever and the increased contractility of the uterus, which diminishes the risks of tropical uterine inertia. They consider that there is far less risk to the mother and fœtus from large doses of quinine than there would be if malaria were untreated. Generally contracted pelvis is very common, but the authors do not favour either induction of premature labour or a trial of labour in doubtful cases. While these methods are applicable to European women, there are certain dangers associated with delivery in India which preclude their routine use. These are summarized as diet deficiency, a 20% hæmoglobin deficiency and a predisposition to infection and obstetric shock, while uterine inertia is very common. For these reasons Cæsarean section is the method of choice in any case presenting difficulties. The chapter on pyrexia in the puerperium is well written, and the authors point out that the clinical picture of puerperal sepsis may be imitated or obscured by some tropical disease dormant for months but showing itself after the disturbance of childbirth. The value of post-natal care is well stressed, and infant feeding has been carefully outlined.

Despite the paucity of illustrations the subject matter is well set out in concise sentences, and the book is a fine record of modern obstetrical practice as applied to India and the tropics.

THE SURGICAL EMERGENCIES OF CHILDHOOD.

Surgical Emergencies in Children. By H. C. EDWARDS, F.R.C.S.; 1936. London: Baillière, Tindall and Cox. Medium 8vo., pp. 274, with 99 illustrations. Price: 12s. 6d.

MR. H. C. EDWARDS has written an excellent little book on surgical emergencies in children. With the exception of the chapters on general anæsthesia and on the ear, nose and throat, the book is based on the author's own experience and practice. It is written clearly and concisely, and correct emphasis is laid on matters calling for particular notice in the emergency surgery of childhood. No subject is treated exhaustively, yet the author manages to convey clear directions on the course of treatment and to say adequately all that need be said on diagnosis and prognosis.

The book does not set out to deal with surgery in childhood as a whole; it is limited to the surgery of emergencies. It is the sort of book which is likely to expand in scope and size with succeeding editions, for it is not always agreed among practitioners what constitutes an emergency; and the admission of fairly urgent surgical diseases, such as congenital pyloric stenosis, will insensibly open the way to the inclusion of other surgical disabilities about which this author could contribute valuable views.

The illustrations are original, not too numerous, and clearly explain what they are supposed to illustrate, though in succeeding editions a little more detail in Figure 49 and Figure 76, or photographs of actual apparatus, would show just where the apparatus should be airtight, and would thus serve as a better guide for the setting up of such apparatus by the uninstructed.

The book should be interesting to the surgeon with experience of children's surgery, and valuable to surgeons whose experience in this branch of surgery is limited.

THE PARATHYREOIDS.

The Parathyroids in Health and Disease. By DAVID H. SHELLING, B.Sc., M.D.: 1935. St. Louis, U.S.A.: The C. V. Mosby Company. Australian Agents: W. Ramsay (Surgical) Proprietary Limited. Royal 8vo, pp. 335, with 26 illustrations. Price: 36s.

THE last decade has seen a very rapid advance in our knowledge concerning the parathyreoids, and we welcome Dr. Shelling's timely review. It is clearly written, well illustrated, and summarizes briefly the established facts concerning the anatomy, physiology and biochemistry of these glands. A bibliography at the end of each section will be found useful to those who desire more detailed information.

Dr. Shelling has not neglected the clinical and pathological aspects of his subject, and the book is particularly useful from these points of view. Nearly half the volume is occupied by a full discussion of tetany and *osteitis fibrosa* in relation to hypo- and hyper-parathyreoidism. The rather shadowy interrelations of other ductless glands with the parathyreoids are also briefly reviewed. The relationship of the parathyreoid to vitamin *D* is adequately discussed. The author suggests that the glands protect the organism from the effects of too prolonged exposure to sunlight or of an excess of vitamin *D*.

The last chapter on the use and misuse of parathyreoid hormone and of parathyreoidectomy is a sound commentary on the application of modern discoveries to the treatment of disease. The author advises the strict limitation of parathyreoidectomy to conditions in which hyper-parathyreoidism can be demonstrated, and justly casts doubt upon its value as a therapeutic measure in

rickets, osteomalacia, renal rickets, renal stone, Paget's disease, *spondylitis deformans*, ankylosing arthritis and scleroderma, in all of which this procedure has been advocated. He emphasizes the inadvisability of attempting to remedy osteoporosis by the production of hypercalcæmia without control of the content of phosphorus in the diet, and points out that excess of calcium may be actively harmful. The use of parathyreoid hormone should be restricted to hypoparathyreoidism, and the author finds its most important application in combating the acute tetany associated with this condition by causing a rapid increase in blood calcium.

In the tetany of pregnancy and lactation, and of rickets and in osteomalacia, cœliac disease and sprue, calcium and vitamin *D* should be given. Parathyreoid hormone is occasionally useful in atrophic conditions, in some cases of œdema of nephritic, nephrotic or cardiac origin, and in acute hepatic insufficiency. In "deleading" in lead poisoning parathyreoid hormone should be given only when the patient is on a diet rich in phosphorus and poor in calcium.

SURGERY OF THE FEMALE PELVIS.

A Textbook of Gynæcological Surgery. By SIR COMYNS BERKELEY and VICTOR BONNEY; 1935. London: Cassell and Company, Limited. Sydney: Angus and Robertson Limited. Royal 8vo, 863 pages, with 530 drawings and 17 colour plates. Price: 67s. 6d.

"A TEXTBOOK OF GYNÆCOLOGICAL SURGERY", written by Sir Comyns Berkeley and Victor Bonney, has won its own reputation in the English-speaking surgical world as a textbook of outstanding merit. The third edition, published in 1935, is a worthy successor to the previous edition, and will place the reputation of this textbook still higher.

In the twenty-four years that have elapsed since the first edition was published there have been many changes in surgery. The ripe experience of the authors, together with their great surgical reputation, is embodied in the edition which has come to hand. It is to all intents and purposes a new book, both as to text and illustration. Indeed, it might be regarded as a general surgical textbook on the lower part of the abdomen in the female, as the authors include very complete descriptions of operations on the intestinal canal, the bladder, the ureter and the rectum.

In the 863 pages which comprise the book the authors have dealt with every possible problem in the surgery of the female pelvis. The illustrations are so clear and simple that they are very easy to follow, and are of as much use to the experienced surgeon as to the beginner. Although a very valuable portion of the text is that which deals with the difficulties and dangers that arise in the various operations described, this is, from a practical point of view, invaluable to the young surgeon, and a very useful reminder to any surgeon, no matter what his experience may be. No surgeon who is interested in the surgery of the female pelvis should be without this very valuable, clear and comprehensive account of the subject.

Post-Graduate Study.

THE SURGICAL UNIT AT THE BRITISH POST-GRADUATE MEDICAL SCHOOL.

IT is now more than a year since Grey Turner, of Newcastle, with Lambert Rogers, of Cardiff, as his chief assistant, took over the direction of the newly established surgical unit in the Hammersmith Hospital. For some time past it has been becoming increasingly apparent that it was not possible to combine successfully

in the existing teaching hospitals of London both undergraduate and post-graduate instruction, and in 1921 a committee appointed by Dr. Addison, then Minister of Health, reported that the existing provision for general post-graduate instruction in London was seriously deficient. The new school came into being as a result of this report and of much subsequent committee work, and in May of last year Their Majesties King George V and Queen Mary formally declared the school open at the Hammersmith Hospital, as this institution had been selected as the most suitable institution with which to associate it. The new school has been recognized as a teaching unit of London University, and has a whole-time staff. On the surgical side the director of the division has been appointed to a professorship in the University and his chief assistant to a readership. Corresponding appointments have been made in medicine, obstetrics and gynæcology, and pathology. The hospital itself is one of the large group of municipal hospitals administered by the London County Council.

Many of those who read this journal have already visited the Post-Graduate School, still in the course of its evolution, and have possibly become familiar with some of the problems connected with it, problems which are inevitably associated with the launching and stabilizing of any such project as that visualized by those responsible for establishing the new school. Already within its first year considerable changes have been made, and will continue to take place. The number of the surgical beds is being increased, and pressure upon these is gradually rising, first because the facilities for treatment are becoming apparent to the practitioners in the neighbourhood, and, secondly, because certain cases of a special character are being transferred to Hammersmith from other hospitals also under the administration of the London County Council. Two first assistants in the surgical unit and four house surgeons were appointed shortly after its establishment, and the numbers of both assistants and house officers are about to be increased.

In October last, on his becoming Professor of Surgery at Cardiff, Lambert Rogers resigned his readership, but still does occasional work in the school, and in October next, A. K. Henry, of Cairo, who has just been appointed, will succeed to the readership.

Facilities for research work are being developed, and the visitor from abroad will find much at the Post-Graduate School to interest him and opportunities for investigating his problems which few other institutions possess. Since London, with its special hospitals, will, no doubt, long remain a Mecca for the surgeon from Australia and New Zealand setting forth on his pilgrimage of surgical revision, the Post-Graduate Hospital, the work at which is chiefly of a general character, will, it is thought, attract him in the first place and become a centre from which and towards which his activities may profitably trend.

Library Notice.

THE Honorary Librarian wishes to announce that the following journals are being regularly received, and are available at the College. Fellows may obtain translations of articles in foreign journals on application.

General Medical and Surgical Journals.

American Journal of the Medical Sciences.
Archives of Physical Therapy.
Australian Journal of Experimental Biology and Medical Science.
Birmingham Medical Review.
British Medical Journal.
Bristol Medico-Chirurgical Journal.
Bulletin of the Johns Hopkins Hospital.
Bulletin of the Neurological Institute of New York.
Calcutta Medical Journal.
Canadian Medical Association Journal.

Clinical Medicine and Surgery.
Collected Papers of the Mayo Clinic.
Edinburgh Medical Journal.
Endocrinology.
Guy's Hospital Reports.
Il Policlinico.
Irish Journal of Medical Science.
Japanese Journal of Experimental Medicine.
La Clinica.
La Presse Médicale.
Medical Journal of Australia.
New England Journal of Medicine.
New York State Journal of Medicine.
New Zealand Medical Journal.
Northwest Medicine.
Proceedings of the Mayo Clinic.
Proceedings of the Royal Society of Medicine.
South African Journal of Medical Science.
South African Medical Journal.
Southern Medicine and Surgery.
St. Bartholomew's Hospital Reports.
The Clinical Journal.
The Lancet.
The Liverpool Medico-Chirurgical Journal.
The Medical Press and Circular.
The Practitioner.
The Quarterly Cumulative Index Medicus.
The Royal Melbourne Hospital Clinical Reports.
Tohoku Journal of Experimental Medicine.
Transactions of the College of Physicians, Philadelphia.

General Surgical Journals.

Acta Chirurgica Scandinavica.
Annals of Surgery.
Archives of Surgery.
Australian and New Zealand Journal of Surgery.
British Journal of Surgery.
Bulletins et Mémoires de la Société des Chirurgiens de Paris.
Der Chirurg.
International Abstract of Surgery.
Journal de Chirurgie.
Lyon Chirurgical.
Surgery, Gynecology and Obstetrics.
Western Journal of Surgery, Obstetrics and Gynecology.

Cancer.

American Journal of Cancer.
Journal of Cancer Research of Sydney.

Chest.

Journal of Thoracic Surgery.

Laryngology and Otology.

Journal of Laryngology and Otology.
The Laryngoscope.

Obstetrics and Gynæcology.

American Journal of Obstetrics and Gynecology.
Journal of Obstetrics and Gynæcology of the British Empire.

Ophthalmology.

Archiv für Augenheilkunde.
Bulletin de la Société Belge d'Ophthalmologie.
Bulletin of the Ophthalmological Society of Egypt.
British Journal of Ophthalmology.
Rassegna Italiana d'Ottalmologia.

Orthopædics

Acta Orthopedica Scandinavica.
Journal of Bone and Joint Surgery.
Revue d'Orthopédie et de Chirurgie de l'Appareil Moteur.

Radiology.

British Journal of Radiology.
Radiology.

Urological Journals.

Archivio Italianò di Urologia.
British Journal of Urology.
Journal d'Urologie.
Journal of Urology.
Urologic and Cutaneous Review.
Zeitschrift für Urologie.

Notices.

PRIMARY FELLOWSHIP EXAMINATION OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND.

THE Council of the College wishes to announce that a primary examination in anatomy and physiology will be held during November-December, 1937, in Melbourne, Sydney and Dunedin, under the following conditions:

The papers will be written simultaneously in all three centres, beginning on Monday, November 29, 1937, and the *viva voce* part of the examination will begin in Melbourne during the same week; in Sydney during the week following that in which the papers are written; in Dunedin during the second or third week after the written papers.

The result will be announced at the termination of the examination in each centre.

The examination is open to candidates who are graduates or undergraduates of the medical faculty of any university recognized by the Royal College of Surgeons of England, and who comply with the conditions of the regulations. (The recognized universities in Australasia are the Universities of Adelaide, Melbourne, Sydney and Dunedin.)

Copies of the regulations, forms of application for admission to the examination, and the schedule of the certificates required may be obtained, free of charge, from the Secretary, the Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

Completed forms of application, together with the certificates required by the regulations, must reach the Secretary of the Royal Australasian College of Surgeons not later than Saturday, May 1, 1937.

The fee for admission to the examination, or reexamination, is £25 sterling, which must be paid to the Secretary of the Royal Australasian College of Surgeons not later than Monday, November 1, 1937.

Candidates who withdraw from the examination, owing to any cause whatsoever, after they have paid their fees will be refunded half the amount of the fee which they have paid.

All inquiries concerning the examination to be addressed to the Secretary, the Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

REGISTRATION OF THE COLLEGE DIPLOMA OF FELLOWSHIP.

THE Council of the College wishes to announce that the New South Wales Medical Board has granted recognition to the College Diploma of Fellowship as a registrable additional qualification.

THE BRITISH POST-GRADUATE MEDICAL SCHOOL.

THE College wishes to draw attention to the announcement of the British Post-Graduate Medical School on page xxxii of the advertisements.

NEW DEVELOPMENTS IN SURGICAL EQUIPMENT.

THE attention of Fellows is drawn to pages xviii and xxvi among the advertisements, which illustrate some recent developments in surgical equipment. The Editorial Committee is responsible for the selection of the equipment illustrated thereon. The publishers will be pleased, whenever possible, to supply the names and addresses of the manufacturers to anyone requiring such information.

Editorial Notices.

EDITORIAL communications should be addressed to the Chairman of the Editorial Committee, 57 Collins Street, Melbourne, or to any member of the Editorial Committee. It is understood that original articles forwarded for publication are offered to THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY solely, unless the contrary be stated.

Reprints can be supplied at cost price; the minimum number is fifty copies. Orders for reprints must be given when the proof is returned.

Exchange journals should be addressed to the Honorary Librarian, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, Victoria, Australia.

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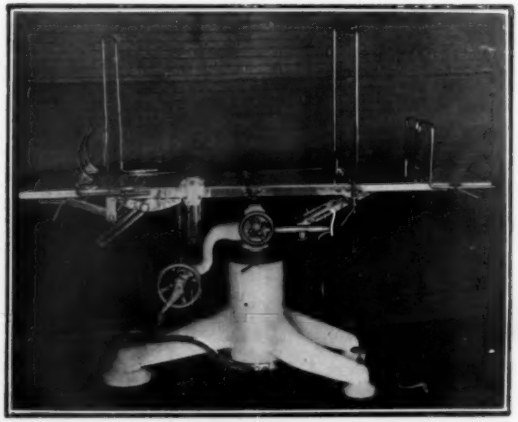

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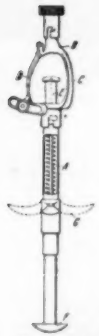

FIGURE 1.

INSTRUMENT FOR DRIVING A KIRSCHNER WIRE.

(*Der Chirurg*, 15th April, 1936, pages 347-348.)

This is an instrument which is used for driving a Kirschner wire through the end of the terminal phalanx close to the base of the nail. Figure 1 shows the details of the instrument: F being the plunger which drives the Kirschner wire, and A being a small spring which causes the plunger to fly back after the wire has been driven through the terminal phalanx.

FIGURE 2.

Figure 2 shows the way in which the thumb is held by the instrument before the Kirschner wire is plunged through it. The wire is then fixed to an apparatus so as to produce extension on the edge.

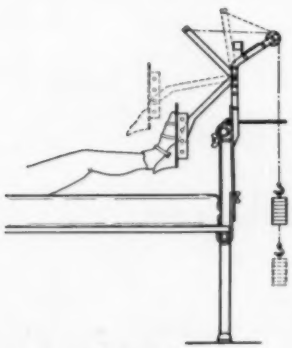

FIGURE 3.

Figure 3 is an arrangement for the mobilization of joints, and especially of the ankle joint. By means of this apparatus, not only can the ankle joint be kept in movement, but also the knee and hip. The picture explains the method.

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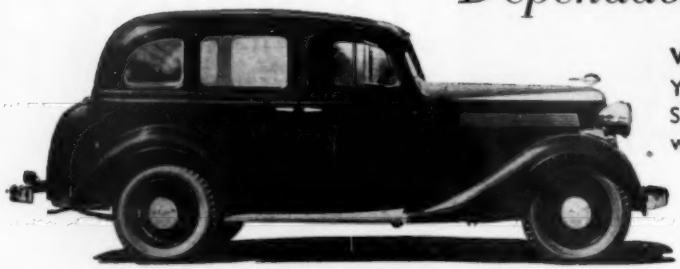

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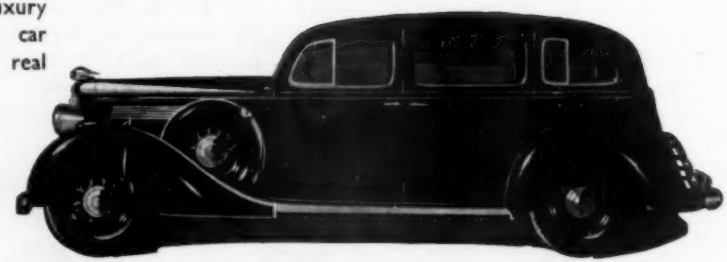

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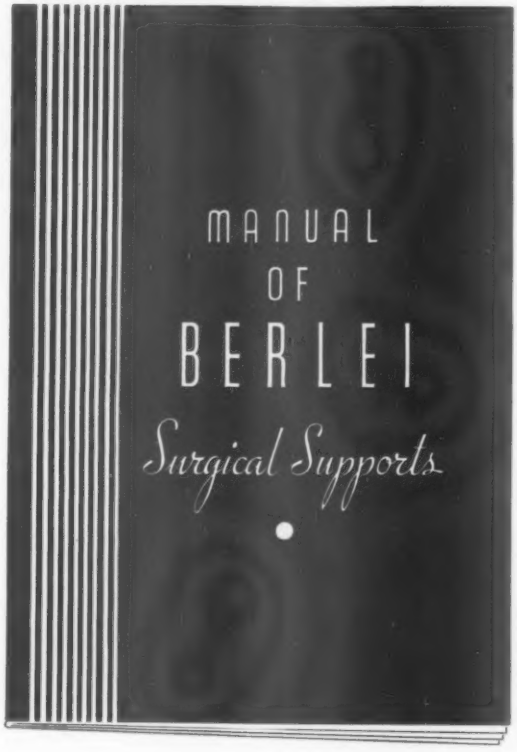

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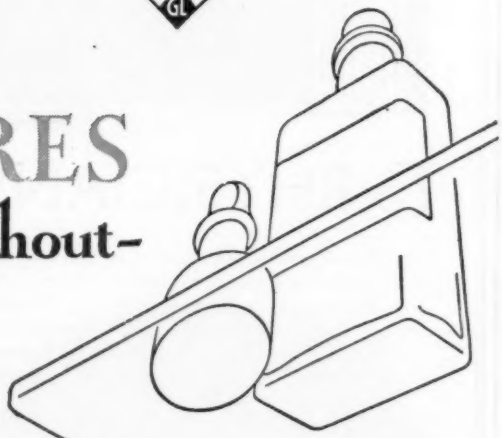

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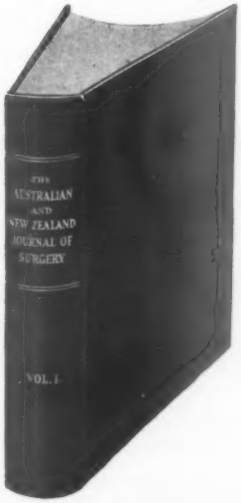

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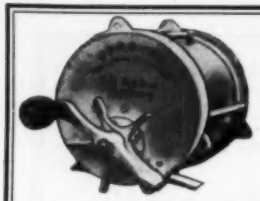

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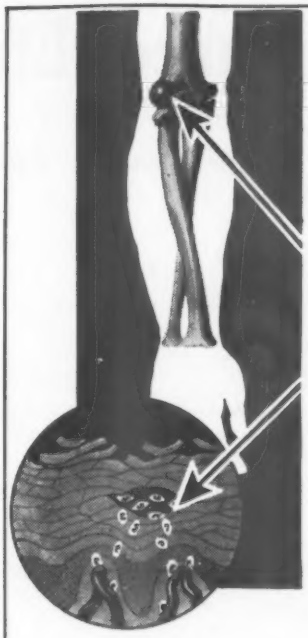

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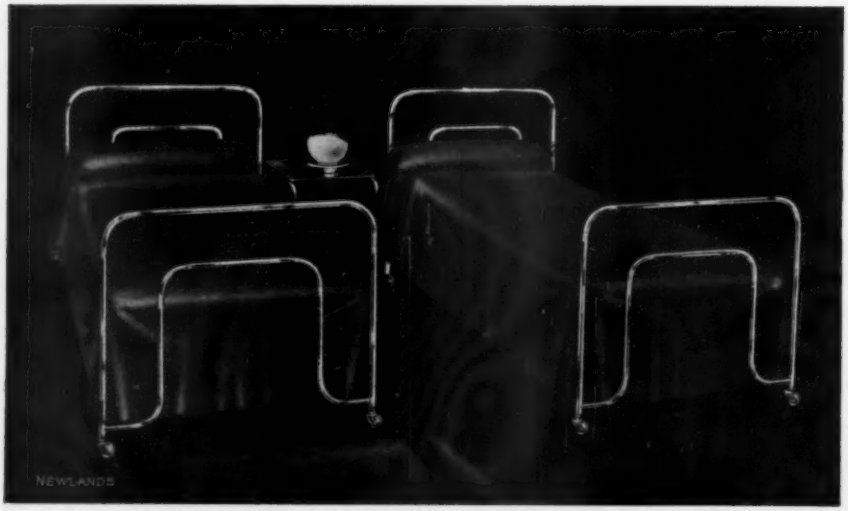

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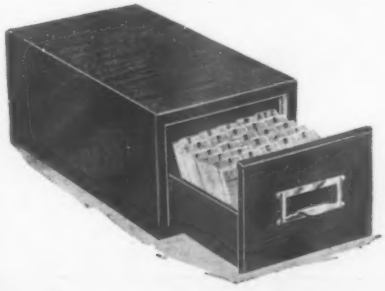

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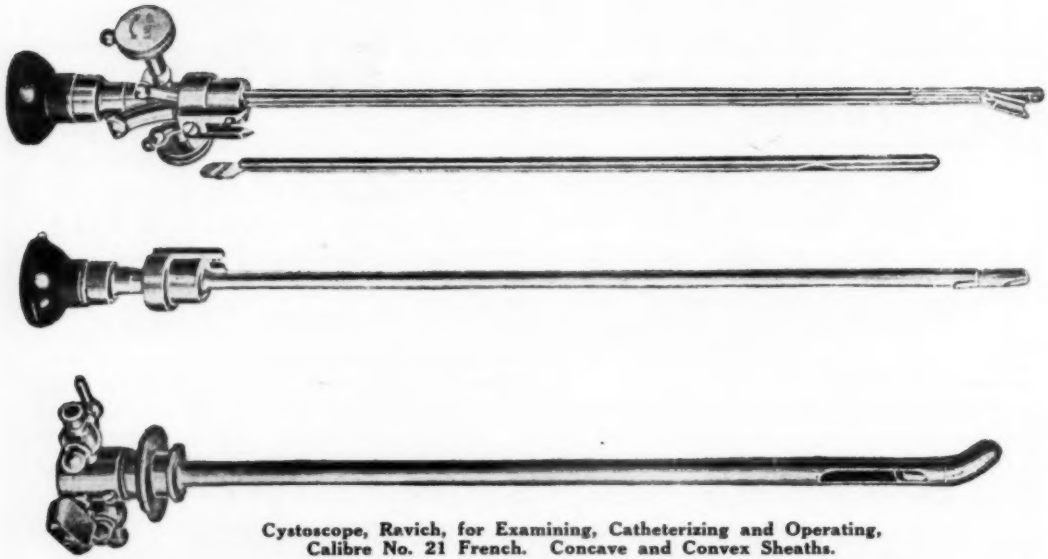

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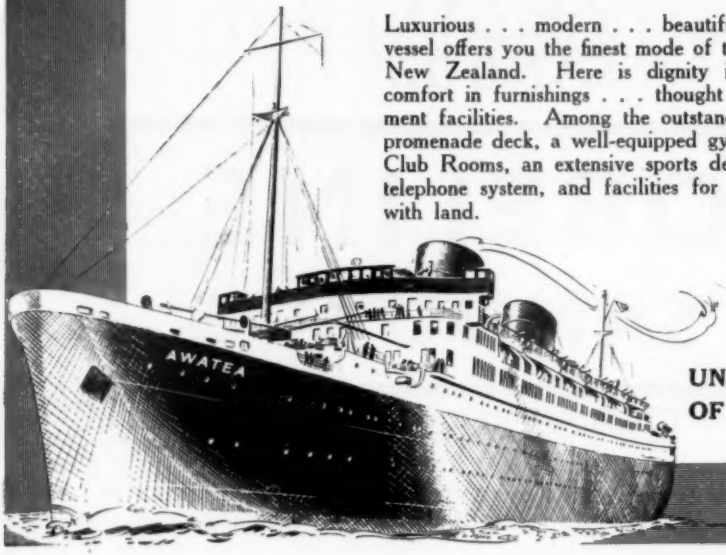

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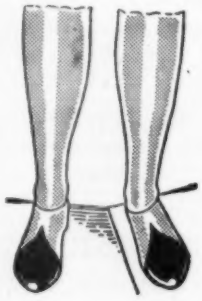

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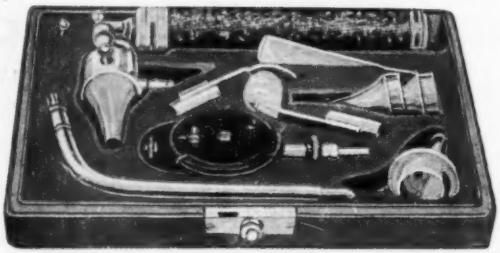

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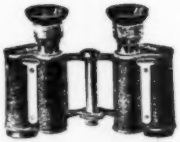

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